TOPOGRAPHIC AND CLINICAL ANALYSIS OF ANTERIOR CIRCULATION STROKES

(A Study of 82 Cases)

Dissertation submitted in partial fulfillment of the requirements for the degree of

D.M. (NEUROLOGY)

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CERTIFICATE

This is to certify that the dissertation entitled *"**TOPOGRAPHIC AND CLINICAL ANALYSIS OF ANTERIOR CIRCULATION STROKES** **(A Study of 82 Cases)"*** was done under our supervision and is the bonafide work of **Dr.K.Bijoy Menon**. It is submitted in partial fulfillment of the requirement for the D.M. (Neurology) examination.

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CONTENTS

INTRODUCTION

Stroke is the most common life threatening neurological disease and the third leading cause of death after heart disease and cancer, accounting for 1 of every 15 deaths. In the elderly, the segment of the population in which stroke occurs most frequently, it is the leading cause of disability requiring long term institutionalization.

Faced with an acute stroke, the physician must determine the cause, estimate the severity, consider the possibility of progression or recurrence and seek ways of stabilizing or reversing it. Investigations should be designed to assist clinicians in subcategorizing patients at three specific levels (1) separating strokes from non strokes such as cerebral tumours and subdrual hematoma (2) distinguishing hemorrhage from infarction and (3) identifying specific pathophysiological sub types of cerebral infarction. Because the possibility of worsening or recurrence is paramount, speedy efforts should be made to arrive at a diagnosis of stroke mechanism using this approach. The ideal test should be inexpensive, noninvasive, accessible, accurate and informative.

In most hospital, the first testing step is an attempt to image the injured site by Computed Tomography CT or magnetic Resonance Imaging MRI. The CT Scan is inexpensive by hospital standards, noninvasive and easily accessible. If, by our observations of CT findings, analyzing the subtle signs and the topographical pattern of brain tissue affection, we are able to reach a reasonable conclusion regarding the type of stroke and its site of origin, it would greatly help us in further management.

In this study, our primary aim has been to identify topographically the different patterns of stroke seen in the anterior circulation of our brain and to look for clues that might point to a specific vascular site. Anterior circulation disease accounts for the bulk of stroke cases and middle cerebral and internal carotid arteries are the main culprits. These are arteries, which are easily accessible by endovascular techniques. In a stroke emergency ward, if the primary physician is reasonably sure by looking at the CT Brain that the disease is in these major arteries, he could order for angiographic studies and plan a neurovascular intervention immediately to clear the narrowed or occluded vessel. Though this approach to an essentially vascular disease is in the future in our country, we think it is in the very

near future and a simple topographical analysis of the CT brain would be a fast and inexpensive first step in the emergency management of this disease.

AIM OF THE STUDY

1. To find out the common anterior circulation stroke patterns based on CT brain.

2. To find out which stroke patterns co-exist with each other.

3. To find out which stroke patterns are associated with disease of the heart.

4. To determine a co-relation between these stroke patterns and their clinical evolution and severity.

REVIEW OF LITERATURE

The anterior circulation of the brain describes the areas of the brain supplied by the right and left internal carotid arteries and their branches. The internal carotid arteries supply the majority of both cerebral hemispheres, except the occipital and medial temporal lobes, which are supplied from the posterior circulation. Ischemic strokes occurring in the anterior circulation are the most common of all ischemic strokes, accounting for approximately 70% of all cases.

The Arteries of the Brain

Since the mode of distribution of the vessels of the brain has an important bearing upon a considerable number of the pathological lesions which may occur in this part of the nervous system, it is important to consider a little more in detail the manner in which the vessels are distributed.

The cerebral arteries are derived from the internal carotid and vertebral, which at the base of the brain form a remarkable anastomosis known as the arterial circle of Willis**.** It is formed in front by the anterior cerebral arteries, branches of the internal

carotid, which are connected together by the anterior communicating; behind by the two posterior cerebral arteries, branches of the basilar, which are connected on either side with the internal carotid by the posterior communicating. The parts of the brain included within this arterial circle are the lamina terminalis, the optic chiasma, the infundibulum, the tuber cinereum, the corpora mammillaria, and the posterior perforated substance.

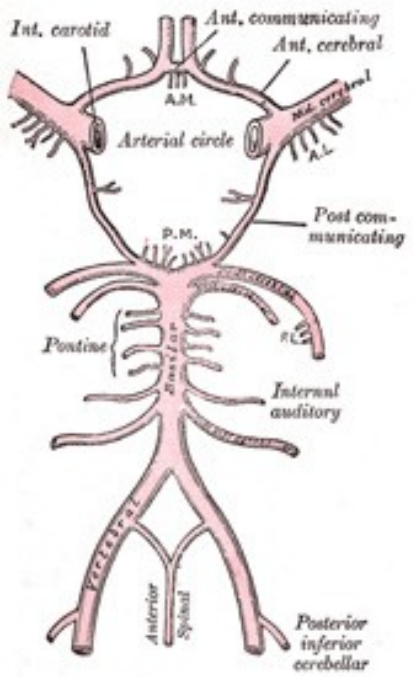


FIG. 1 Diagram of the arterial circulation at the base of the brain. A.L. Antero-lateral. A.M. Antero-medial. P.L. Postero-lateral. P.M. Posteromedial ganglionic branches.

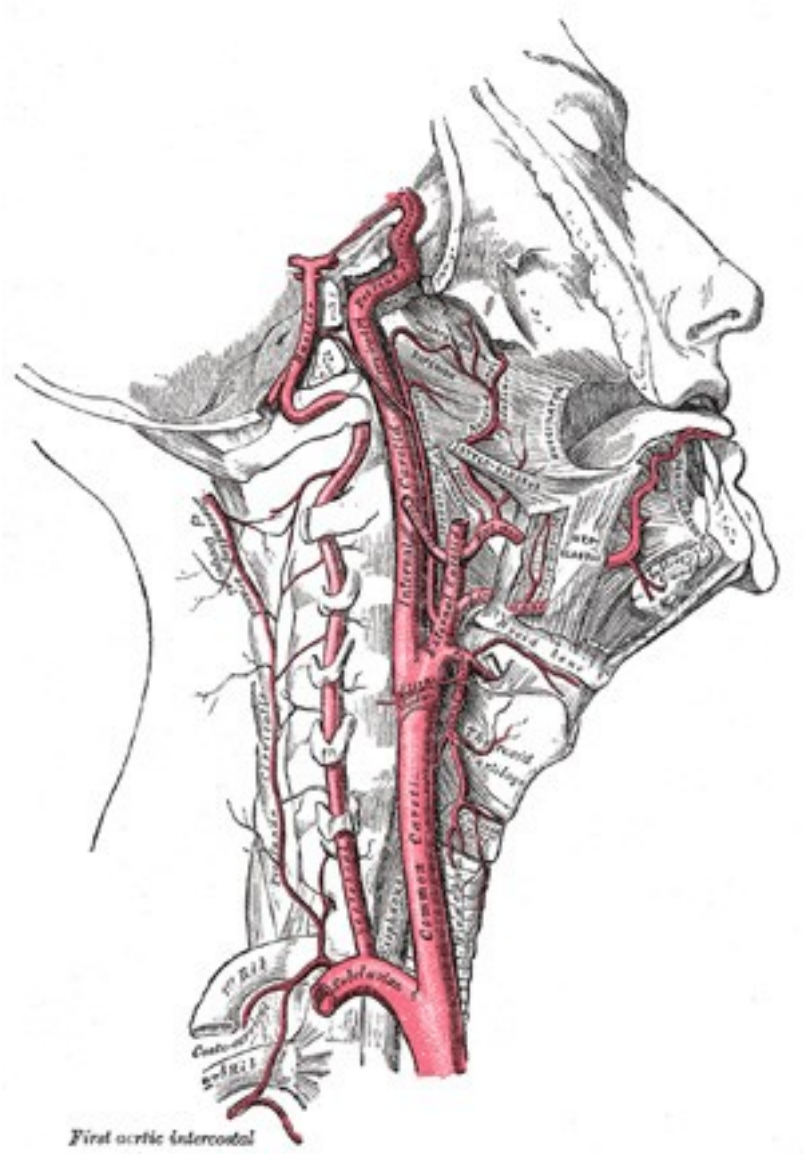


Fig.2 Anatomy of the Internal Carotid Artery

The Ganglionic System.-All the vessels of this system are given off from the arterial circle of Willis, or from the vessels close to it. They form six principal groups: (I) the antero-medial group, derived from the anterior cerebrals and anterior communicating; (II) the postero-medial group, from the posterior

cerebrals and posterior communicating; (III and IV) the right and left antero-lateral groups, from the middle cerebrals; and (V and VI) the right and left postero-lateral groups, from the posterior cerebrals, after they have wound around the cerebral peduncles. The vessels of this system are larger than those of the cortical system, and are what Cohnheim designated terminal arteries - that is to say, vessels which from their origin to their termination neither supply nor receive any anastomotic branch, so that, through any one of the vessels only a limited area of the thalamus or corpus striatum can be injected, and the injection cannot be driven beyond the area of the part supplied by the particular vessel which is the subject of the experiment.

The Cortical Arterial System

The vessels forming this system are the terminal branches of the anterior, middle, and posterior cerebral arteries. They divide and ramify in the substance of the pia mater, and give off branches which penetrate the brain cortex, perpendicularly. These branches are divisible into two classes, long and short. The long, or medullary arteries, pass through the gray substance and penetrate the subjacent white substance to the depth of 3 or 4

cm., without intercommunicating otherwise than by very fine capillaries, and thus constitute so many independent small systems. The short vessels are confined to the cortex, where they form with the long vessels a compact net-work in the middle zone of the gray substance, the outer and inner zones being sparingly supplied with blood. The vessels of the cortical arterial system are not so strictly “terminal” as those of the ganglionic system, but they approach this type very closely, so that injection of one area from the vessel of another area, though possible, is frequently very difficult, and is only effected through vessels of small caliber. As a result of this, obstruction of one of the main branches, or its divisions, may have the effect of producing softening in a limited area of the cortex.

Collateral blood supply of the brain

Fields described this in 1985. The actual pattern of collateral blood flow depends on where the major vessels are stenosed or occluded and on which collateral channels are available and free from disease.

On the whole, the development of collateral channels is more effective if the major vessel occlusion occurs over weeks or

months rather than suddenly. Collateral blood flow may develop via:

A. Extracranial connections:

- In the orbit, branches of ECA anastomose with branches of ophthalmic artery.
- Branches of ECA anastomose with branches of vertebral artery.
- Branches of vertebral artery anastomose with branches of subclavian artery.
- Branches of ECA anastomose with branches of subclavian artery.

B. Intracranial connections:

Circle of Willis

It is a network of blood vessels present at the base of the brain. This polygon of blood vessels is formed by the proximal parts of the two anterior cerebral arteries connected by the ACoA and the proximal parts of the two posterior cerebral arteries connected to the distal internal carotid arteries by the posterior communicating arteries. However fifty per cent of circles have

hypoplastic or absent segments and the potential for collateral flow is not always as good as it might first appear.

Leptomeningeal anastomoses:

It lies on the surface of the brain. They develop between the cortical branches of the anterior, middle and posterior cerebral arteries.

Dural anastomoses

It occurs between meningeal branches of the ICA, ECA and the vertebral arteries.

Anterior choroidal artery

It is a branch of ICA that can anastomose with the posterior choroidal artery

Posterior choroidal artery

A branch of ECA.

PHYSIOLOGY OF THE CEREBRAL CIRCULATION

A. Blood Brain Barrier

This barrier insulates the brain and its extracellular fluid, including the cerebrospinal fluid (CSF), from many of the body's blood borne chemical perturbations, such as circulating drugs, immunogenic antigens and electrolyte changes. The anatomic barrier lies in the intracranial endothelium, where tight intracellular junctions weld the entire inner vascular surface into a continuous membranous sheet. As a result, only nonpolar materials that have a small molecular size, are lipid soluble or are transported across the membrane by specific carrier systems or pumps transgress the endothelium with any rapidity.

Transient breaches of the barrier occur under a variety of circumstances but have little ill effect on brain function. Sustained, partial barrier alterations occur in areas of cerebral neoplasms, inflammation or edema associated with such conditions. Severe damage to barrier transport mechanisms can intensify brain infarction during ischemia.

B. Regulation of cerebral blood flow

Cerebral blood flow (CBF) in man is about 50 ml / 100 g of brain / minute. It has been shown that CBF, cerebral blood volume (CBV) and cerebral energy metabolism measured as cerebral metabolic rate of oxygen (CMRO2) or of glucose (CMRglu) are all coupled and higher in gray than white matter. This means that the oxygen extraction fraction (OEF) remains about the same (approximately forty per cent) throughout the brain, therefore, in normal resting human brain, CBF (i.e. flow) is a reliable reflection of CMRO2 (i.e. function).

CBF depends on cerebral perfusion pressure (CPP) and cerebrovascular resistance. The perfusion pressure is the difference between systemic arterial pressure and venous pressure at exit of the subarachnoid space, the latter being approximated by the intracranial pressure.

Autoregulation

It is a characteristic of the brain to adjust its own blood supply. In normal individuals, CBF remains constant when the mean arterial pressure is between 60 and 160 mmHg which, in normal

circumstances, when the intracranial venous pressure is negligible, is the same as the CPP. Whether myogenic, metabolic or neurogenic processes are responsible for this process is unknown. Autoregulation is impaired or abolished in damaged areas of the brain (e.g. by ischemia, trauma, etc.) so that CBF becomes pressure passive and follows perfusion pressure.

Pathophysiology:

Ischemic strokes in the anterior circulation are caused most commonly by occlusion of one of the major intracranial arteries or of the small single perforator (penetrator) arteries. The most common causes of arterial occlusion involving the major cerebral arteries are (1) emboli, most commonly arising from atherosclerotic arterial narrowing at the bifurcation of the common carotid artery, from cardiac sources, or from atheroma in the aortic arch and (2) a combination of atherosclerotic stenosis and superimposed thrombosis. Lacunar strokes are believed to be caused by lipohyalinotic intrinsic disease of the small penetrating vessels.

The most common sites of occlusion of the internal carotid artery are the proximal 2 cm of the origin of the artery and,

intracranially, the carotid siphon. Factors that modify the extent of infarction include the speed of occlusion and systemic blood pressure. Occlusion of the internal carotid artery is not infrequently silent, because external orbital-internal carotid and willisian collaterals can open up if the occlusion has occurred gradually over a period of time. Mechanisms of ischemia resulting from internal carotid artery occlusion are, most commonly, artery-to-artery embolism or propagating thrombus and perfusion failure from distal insufficiency.

The MCA is the largest of the intracerebral vessels and supplies through its pial branches almost the entire convex surface of the brain, including the lateral frontal, parietal, and temporal lobes; insula; claustrum; and extreme capsule. The lenticulostriate branches of the MCA supply the basal ganglia, including the caput nuclei caudati, the putamen, the lateral parts of the internal and external capsules, and sometimes the extreme capsule. Occlusion of the MCA commonly occurs in either the main stem (M1) or in one of the terminal superior and inferior divisions (M2). Occlusion of the M1 segment of the MCA prior to the origin of the lenticulostriate arteries in the presence of a

good collateral circulation can give rise to the large striatocapsular infarct.

Occlusion of the MCA or its branches is the most common type of anterior circulation infarct, accounting for approximately 90% of infarcts and two thirds of all first strokes. Of MCA territory infarcts, 33% involve the deep MCA territory, 10% involve superficial and deep MCA territories, and over 50% involve the superficial MCA territory.

The ACA supplies the whole of the medial surfaces of the frontal and parietal lobes, the anterior four fifths of the corpus callosum, the frontobasal cerebral cortex, the anterior diencephalon, and the deep structures. Occlusion of the ACA is uncommon, occurring in only 2% of cases, often through atheromatous deposits in the proximal segment of the ACA.

The anterior choroidal artery supplies the lateral thalamus and posterior limb of the internal capsule. Occlusion of the anterior choroidal artery occurs in fewer than 1% of anterior circulation strokes. Often, ischemia in the distribution of the ophthalmic artery is transient in the setting of symptomatic internal carotid artery occlusion (ie, transient monocular

blindness, occurring in approximately 25% of patients), but central retinal artery ischemia is relatively uncommon, presumably because of the efficient collateral supply.

Occlusion of single penetrating branches of the middle and anterior cerebral arteries that supply the deep white and gray matter produce the lacunar type of stroke. These occlusions account for as many as 20% of ischemic strokes.

Borderzone (BZ) infarcts or watershed infarcts are ischemic lesions that occur in the junction between two main arterial territories. These account for approximately 10% of all brain infarcts. Supratentorial BZs consist of three regions: 1) the anterior BZ, the superficial area between the middle cerebral artery (MCA) and anterior cerebral artery (ACA); 2) the posterior BZ, the superficial area between the territories of the MCA and the posterior cerebral artery (PCA); and 3) the internal BZ, the deep area between the territories of the cortical branches and the penetrators of the MCA. Haemodynamic theory has been emphasized as the most predominant mechanism of BZ infarction; it states that mild-to-moderate hypotension selectively diminishes perfusion in the BZs in patients with major cerebral

artery occlusive disease. Recently, several investigators have reported that many BZ infarcts cannot be explained by the haemodynamic theory and they are supposed to be embolic; i.e., emboli of a certain small size occlude terminal portions of the cortical branches and cause superficial watershed infarction. These conflicting observations indicate the need to reexamine the pathogenesis of BZ infarcts. Topographically, the internal borderzone is in the region of the corona radiata for the superficial perforating and deep branches of the MCA and in the region of the centrum semiovale for the deep branches of the MCA and ACA. It is the shape of these infarcts that differentiate a haemodynamic from an embolic origin. A linear/confluent rosary like shape favors haemodynamic mechanism whereas discrete infarcts favor embolic/lacunar mechanism[1,2,3,4,5,12]

The acute ischemic process varies markedly from patient to patient. Patients with similar clinical syndromes may have markedly different pathophysiological profiles. Many new pathophysiological insights have been obtained from studies using functional brain imaging (eg, magnetic resonance imaging [MRI], positron emission tomography [PET], single-photon emission computed tomography [SPECT]). Several

pathophysiological ischemic stroke syndromes can be identified on the basis of imaging parameters of perfusion and tissue injury that could be used to target stroke treatment. Using new MRI methods, the following 3 patterns have been identified:

- Perfusion-diffusion mismatch, which may represent a situation of viable but ischemic tissue that could be salvaged by timely reperfusion. In this pattern, a larger area of hypoperfusion surrounds a zone of ischemic injury on diffusion-weighted imaging. This pattern occurs in approximately 70% of patients in the first 24 hours.
- Complete ischemia, in which the perfusion and diffusion lesions are of equivalent size, likely representing a complete infarct. This pattern has been identified in approximately 10-20% of patients in the first 24 hours.
- Reperfusion pattern, in which a perfusion deficit no longer exists. This pattern occurs in approximately 10-15% of patients in the first 24 hours.

Efforts are now underway to incorporate MR angiography findings as well.

Reperfusion is an important part of the ischemic process, and by 24 hours, 20-40% of arterial occlusions have begun to clear, with recanalization rates of 70% by 1 week and 90% by 3 weeks. Early reperfusion (<24 h) may have significant prognostic benefits and is associated with improved outcome and smaller infarct size, but later reperfusion may not alter outcome significantly and may be associated with hemorrhagic conversion of the infarct and edema formation.

Frequency of anterior circulation stroke:

- Approximately 731,000 new and recurrent cases of stroke occur each year in the United States. 80% of these are ischemic strokes. Anterior circulation ischemic stroke accounts for approximately 70% of all ischemic strokes. Approximately 409,360 new cases of anterior circulation ischemic stroke per year are reported in the United States.

- Internationally: The risk of stroke is highest in Eastern Europe, followed by Western Europe, Asia, the rest of Europe, and North America.

Mortality/Morbidity:

- Stroke is the third leading cause of death in the United States and the leading cause of adult disability. High rates of morbidity and mortality are associated with all types of ischemic strokes, but the prognosis varies among subtypes. For example, the lacunar syndromes (ie, caused by occlusion of a single small penetrating artery) quite often are associated with a good prognosis and have a better prognosis than MCA syndromes.

- Overall, at 6 months after a stroke, as many as 30% of patients have died, 20-30% are moderately to severely disabled, 20-30% have mild to moderate disability, and 20-30% are without deficits.

- Stroke recurs in as many as 10% of stroke survivors in the first 12 months after stroke, with an incidence of 4% per year thereafter. After transient ischemic attack, the risk of stroke is 10.5% over the next 3 months, with the highest risk in the 2 days following TIA.

Race: The patterns of arterial occlusion are different in African Americans and Asians than in Caucasians.

- Asians and African Americans have higher rates of intracranial arterial occlusive disease than Caucasians. The intracranial arterial occlusive disease in these populations typically involves the main stem of the MCA or the ACA.

- In Caucasians, the arterial occlusive disease typically involves the extracranial carotid arteries, and lesions in the middle and anterior cerebral arteries are usually of embolic origin.

Sex: Strokes at all ages are more likely to occur in men, but overall more strokes occur in women. This is because strokes occur more commonly at older ages and females have a longer life span than males (the native protective effect of estrogen is lost at menopause). This disparity may become greater in the future with the aging of the population.

Age: The incidence of stroke rises exponentially with age, particularly in individuals older than 55 years.

- However, 25% of all strokes occur in individuals younger than 65 years of age; so stroke is not just a condition of the elderly.
- Strokes can occur at any age.

History: Patients typically present with sudden onset of focal neurological symptoms. Specific features of the time course and evolution, focal neurological symptoms, and global symptoms are listed below.

Time course and evolution

- Sudden or rapid onset
- Reaches maximal intensity within 24 hours
- Gradual or stepwise worsening in as many as 30% of patients

Focal neurological symptoms

- Cognitive impairment - Difficulty with speech
- Weakness or incoordination - Unilateral
- Numbness or loss of sensation, typically unilateral

- Dysarthria
- Visual loss, either in one eye or in one visual field.

Global symptoms

- Headache
- Altered mental status
- Syncope
- Seizure
- Coma

Physical:

Left hemisphere (ie, dominant)

- Right hemiparesis, variable involvement of face and upper and lower extremity
- Right-sided sensory loss, in a similar pattern to the motor deficit; usually involves all modalities, decreased stereognosis, graphesthesia
- Right homonymous hemianopia
- Dysarthria

- Aphasia, fluent and nonfluent
- Alexia
- Agraphia
- Acalculia
- Apraxia of the left limbs

Right hemisphere (ie, nondominant)

- Left hemiparesis (same pattern as on right)
- Left-sided sensory loss (similar pattern as the motor deficit)
- Left homonymous hemianopia (same pattern as on right)
- Dysarthria
- Neglect of the left side of environment
- Anosognosia
- Asomatognosia
- Loss of prosody of speech
- Flat affect

Cortical and subcortical: Findings consistent with both cortical and subcortical localization can be seen in this clinical scenario.

ACA territory

- Crural paresis > arm paresis
- Frontal signs (eg, abulia)

Anterior choroidal artery territory

- Hemiparesis
- Hemianesthesia
- Homonymous hemianopia

Lacunar syndromes

- Pure motor hemiparesis
  - Contralateral, usually affecting the face and upper and lower extremities equally
  - Also associated with dysarthria
  - No sensory or visual loss or cognitive impairment
- Pure sensory stroke

        - Contralateral loss of all sensory modalities, equally affecting the face and upper and lower extremities
        - No motor signs, dysarthria, visual loss, or cognitive impairment

    - Dysarthria-clumsy hand syndrome - Dysarthria, dysphagia, contralateral tongue and facial weakness and paresis, and clumsiness of the contralateral arm and hand

    - Homolateral ataxia and crural paresis - Paresis of the contralateral leg and side of the face, prominent ataxia of the contralateral leg and arm—also called "ataxic hemiparesis," meaning ataxia and weakness on the same side.
    - Isolated motor/sensory stroke
        - Paralysis and sensory loss of the contralateral leg, arm, and face
        - No visual loss or cognitive impairment

Causes: Risk factors include epidemiologic risk factors (ie, not modifiable) and potentially modifiable risk factors.

Epidemiologic risk factors

- Age (risk rises exponentially with age)
- Sex (more common in males at all ages)
- Race (African American > Asian > Caucasian)
- Geographic (Eastern Europe > Western Europe > Asia > rest of Europe or North America)
- Genetic risk factors (stroke or heart disease in individuals younger than 60 y; some familial syndromes, eg, cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy [CADASIL])

Potentially modifiable risk factors

- Hypertension (diastolic or isolated systolic)
- Diabetes mellitus type 1 or 2
- Atrial fibrillation
- Smoking

- Coronary artery disease
- Hypercholesterolemia
- Alcohol abuse
- Drug abuse (eg, cocaine)
- Oral contraceptive use
- Pregnancy

Lab Studies:

- The following laboratory tests are indicated in the patient with stroke both to assist in their acute care and to uncover any underlying medical conditions that could complicate the clinical course.
  - Coagulation profile
  - Glucose
  - Electrolytes
  - Liver function tests
  - Erythrocyte sedimentation rate (ESR)
  - Complete blood count (CBC)

Imaging Studies:

- Brain CT scan
    - Noncontrast CT scan of brain is required emergently to rule out cerebral hemorrhage, subdural hematoma, and other intracerebral pathology prior to the administration of thrombolytic therapy.
    - Early signs of infarction that can be detected with CT scan include loss of gray-white matter differentiation and cortical sulcal effacement. The hyperdense MCA sign is indicative of thrombus in the MCA.
    - Topography of infarcts in a CT Brain helps in indicating the vessel involved and the pathophysiology.
    - Other advances in CT scan include the advent of CT angiography and CT perfusion imaging.
- MRI
    - New MR sequences such as diffusion-weighted imaging (DWI) allow detection of ischemic lesions within minutes of stroke onset. Lesions appear as

hyperintense and are easily distinguishable from the surrounding brain.

- Even very small lesions can be detected, and old lesions may be distinguished from new ones by measuring the apparent diffusion coefficient.
- In combination with MR angiography (MRA) and MR perfusion imaging, this modality allows multiple aspects of the ischemic process to be identified in a scanning session of approximately 25 minutes. This is available in many tertiary referral centers.

- Transcranial Doppler ultrasound
  - Transcranial Doppler is used for rapid and noninvasive identification of the site of major arterial occlusion in the MCA, internal carotid artery, and ACA.
  - It also is used to identify embolic load with emboli detection.
- Chest x-ray - Used to determine heart size and pulmonary status

Other Tests:

- Cardiac echocardiography
    - Cardiac echocardiography helps in ruling out a cardiac source of cerebral embolism and in identifying aortic arch atheroma.
    - Transesophageal echocardiography is the investigation of choice, as it has higher detection rates for lesions in the left atrium (eg, thrombus) and the aortic arch.
- Imaging of the neck vessels
    - Imaging of the neck vessels helps in ruling out a significant carotid artery stenosis as a cause of stroke that may require surgical intervention.
    - Perform imaging with ultrasound, MRA, or conventional digital subtraction angiography.
- Hypercoagulability screen - For patients with cryptogenic stroke and a possibility of a hypercoagulable etiology
- ECG

MATERIAL AND METHODS

Patients : Between September 2004 and August 2005, patients admitted in our hospital with stroke were assessed by CT brain for Anterior Circulation infarcts. The patients came from an extensive catchment area in and around Chennai and were mainly from a lower socioeconomic strata. 82 patients fitting the following inclusion and exclusion criteria were enrolled in the study.

Inclusion criteria :

1. Patients admitted with stroke in our hospital with CT brain showing evidence of Anterior circulation involvement.
2. Onset of stroke symptoms within the last one month of admission.

Exclusion criteria:

1. Age < 12 years
2. Isolated lacunal infarts of size > 1.5cm
3. Neuroradiological evidence of either intracerebral hemorrhage or venous infarcts

4. Clinical or investigatory evidence of demyleinative, neoplastic, traumatic, inflammatory, infective or degenerative pathology.

There were 64 males and 18 females in the study. The oldest person was 82 years and the youngest was 20 years of age.

The CT Brains were analysed based on the templates given by Damasio. These are shown at the end of this section. The CT Brains were then assigned to six patterns as elucidated by Szabo[1]. These are described here along with pertinent definitions. A pictorial description is given at the end of this section.

I. Pattern 1 or large territorial infarction. This means a hypodense area in the superficial vascular territory of a main cerebral artery, in this case middle cerebral (MCA) and anterior cerebral artery (ACA).

Pattern1 is subdivided into

a) Partial MCA infarction (occlusion of either the superior or inferior division of MCA)

b) Large MCA infarction (involvement of at least 2 of 3 areas supplied by the MCA i.e. superior, inferior and deep).

c) Total MCA infarction (proximal occlusion at bifurcation on trifurcation with absence of efficient collaterals with involvement of all 3 areas supplied by the MCA).

d) Complete ACA + MCA infarction – Total MCA infarction + ACA infarction.

II. Pattern 2 – This is a subcortical lesion involving the striatocapsular region above or with corona radiate involvement too.

It is an infarct in the territory of the deep perforating branches of the internal carotid artery (ICA) or MCA trunk and is assumed to be due to occlusion of MCA in the presence of patent leptomeningeal collaterals or due to emboli into the deep perforators of the ICA and MCA.

Pattern 2 is subdivided into

a. Large striatocapsular infarcts > 2 cm in size. These are assumed to be due to occlusion at the M1 segment of the MCA with good leptomeningeal collaterals.

b. Lacunar infarcts of size < 2 cm in the striatocapsular region. Isolated single lesions were excluded from the study.

c. Discrete corona-radiata infarcts involving the striato capsular region too.

Discrete corona-radiata infarcts are isolated, non contigous infarcts of size > 1.5cm in the paraventricular regions.

III. Pattern 3 – This means a large lesion involving the cortex with additional smaller cortical or subcortical lesions. This is assumed to be due to partial fragmentation of embolus in a large artery ie the proximal MCA or ICA.

IV. Pattern 4 – This means several disseminated small lesions in random fashion in the distal territory of the MCA, involving mainly cortical regions but also some subcortical regions.

This is due to fragmentation of an embolus or multiple microemboli in smaller vessels.

V. Pattern 5 – Lesions in the border zones or haemodynamic risk zones. These are divided into.

a) Cortical borderzone infarcts – these occur between 2 or 3 main cerebral arteries and the arterial territory divides the infarct into equal parts.

The anterior cortical border zone and posterior cortical borderzone are shown in the templates of Damasio given at the end of this section.

b) Subcortical borderzone or terminal zone infarcts

These are linear, rosary like, confluent hypodense areas in the border between the deep and superficial perforating arteries of the MCA in case of corona radiata infarcts and between the perforating arteries of ACA and MCA in case of centrum semiovale infarcts.

The **centrum semiovale** is the region of white matter in the supraventricular region whose outermost limit is the cortical ribbon and innermost limit is the corona radiata.

VI. Pattern 6 – Discrete hypodensities in the region of the corona radiate or centrum semiovale.

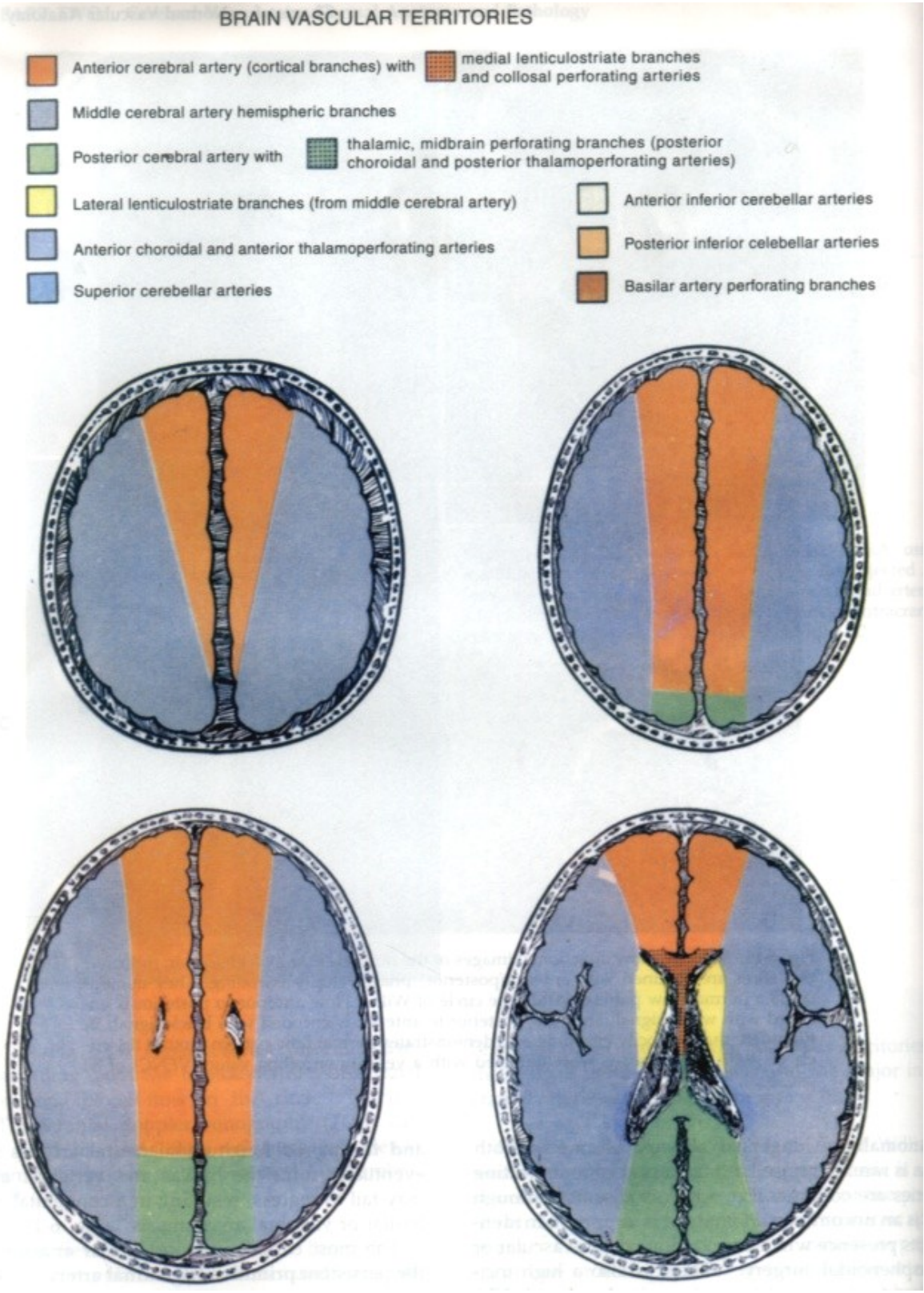
The discrete hypodensities in the coronaradiata region are extensions of lenticulostriate / deep perforator artery infarcts.

Discrete hypodensities in the centrum semiovale are due to infarcts of the superficial perforators which are medullary perforating branches of the pial leptomeningeal MCA branches.

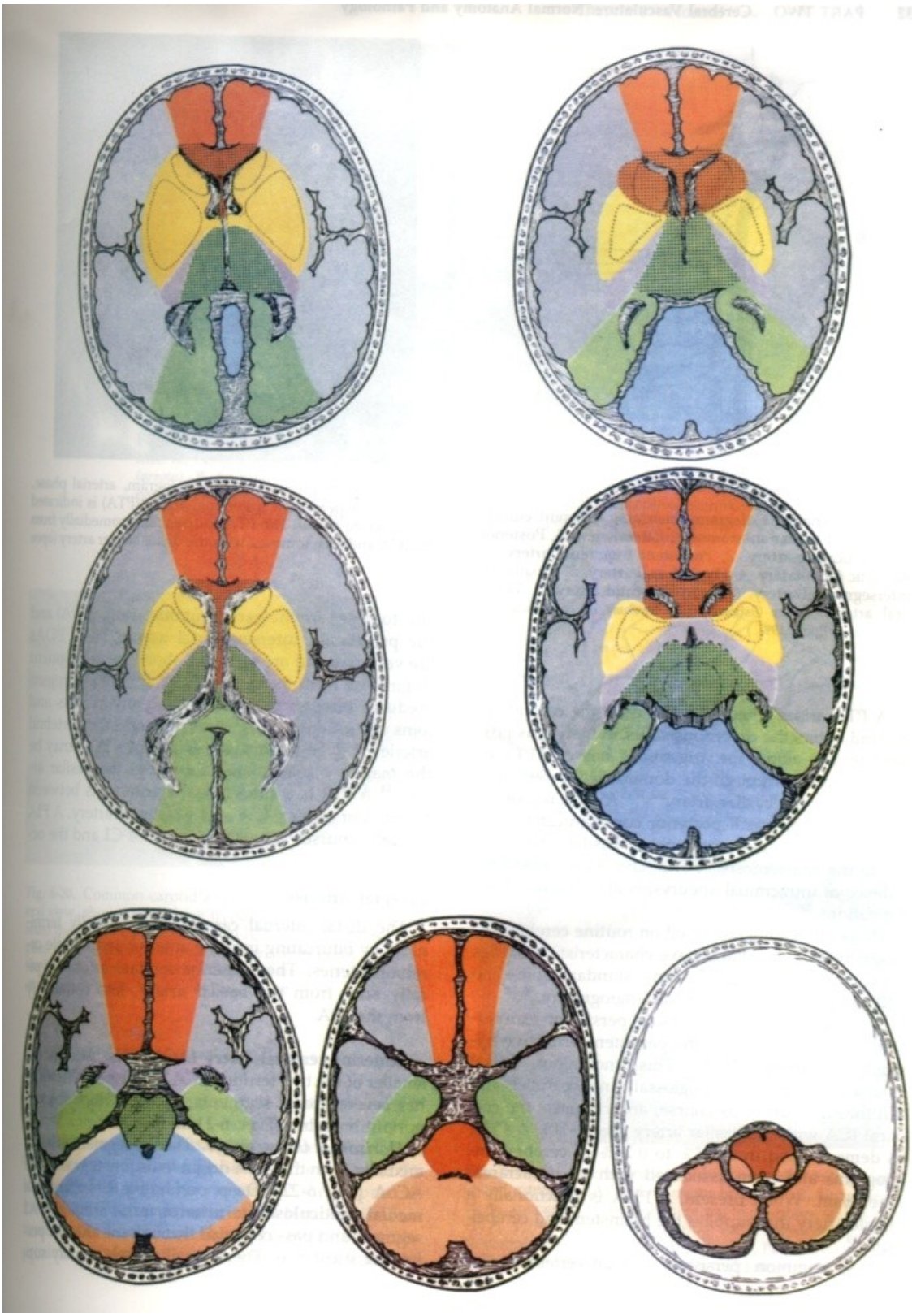
Infarcts which did not fit if any of these patterns were considered “unclassified”.

The patients were assessed clinically with a detailed history and examination after admission. All patients underwent routine blood and urine investigations, ECG, echocardiography and some underwent extracranial Doppler studies and MRI / MRA studies. The details were entered in a proforma and tabulated.

The results were analysed and are presented.



DAMASIO'S TEMPLATES WITH LEGENDS



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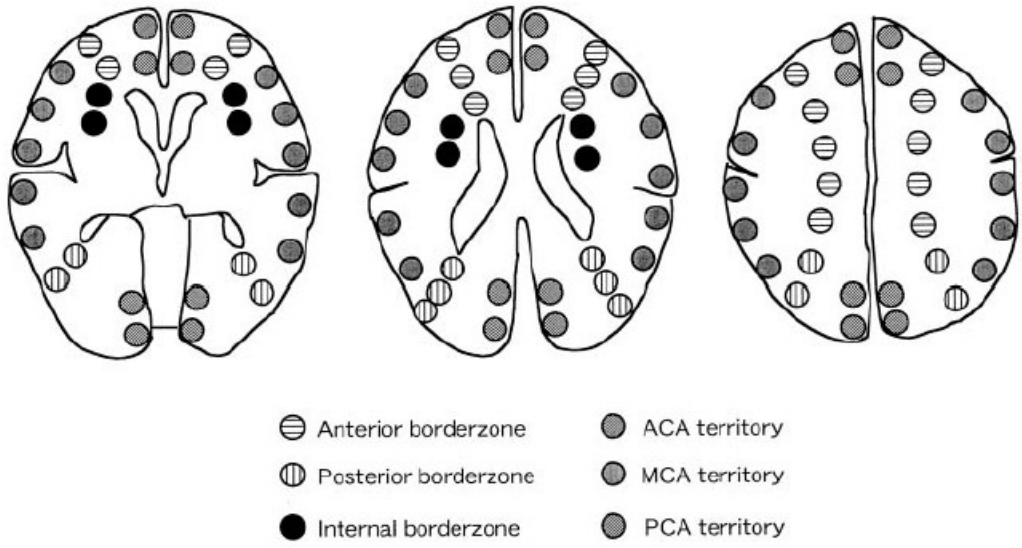
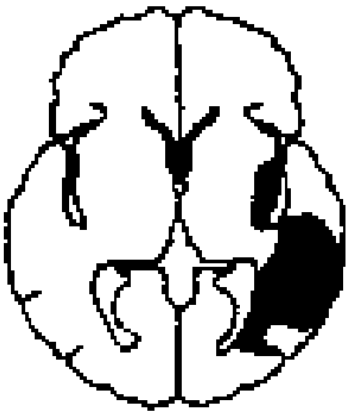
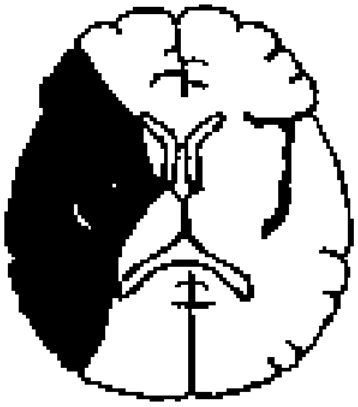


DAMASIO'S TEMPLATES WITH LEGENDS

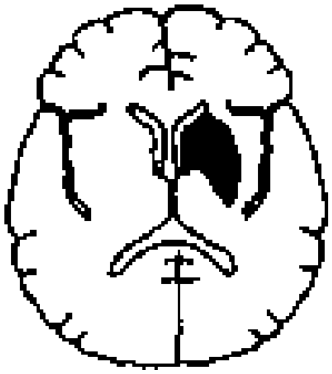
PATTERNS OF ANTERIOR CIRCULATION INFARCTS



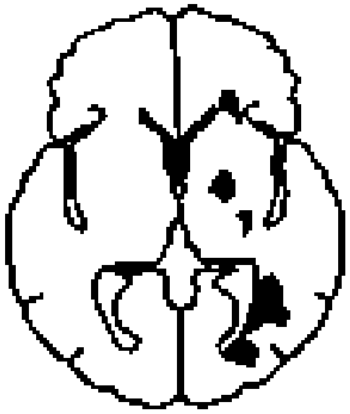
Pattern 1 Partial MCA infarct



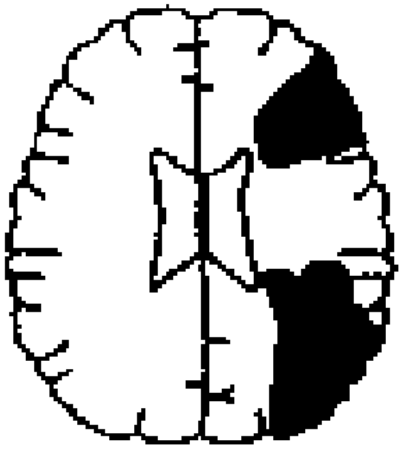
Pattern 1 total MCA infarct



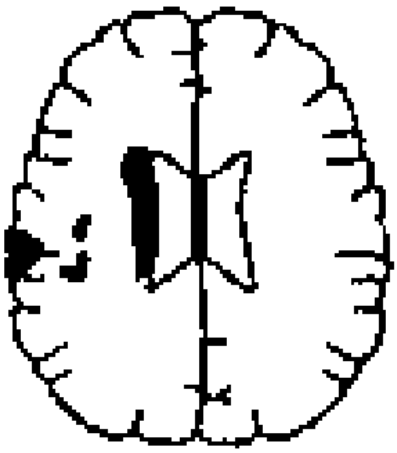
Pattern 2 Large striatocapsular infarct



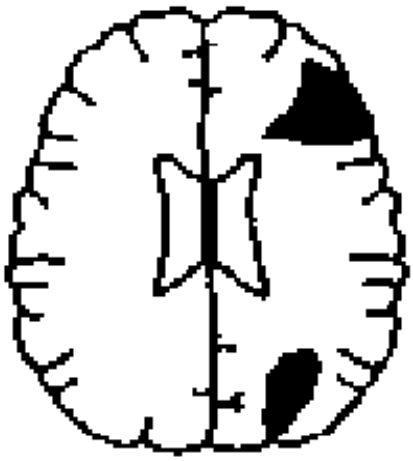
Pattern 2 lacunar striatocapsular infarcts



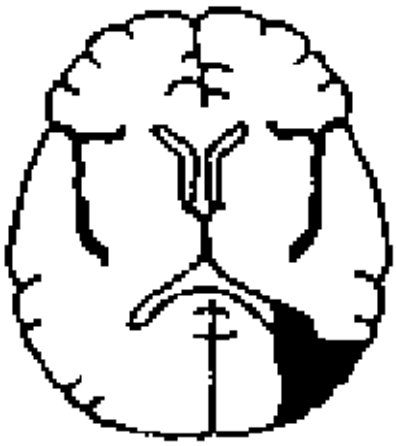
Pattern 3 large fragmentary infarct



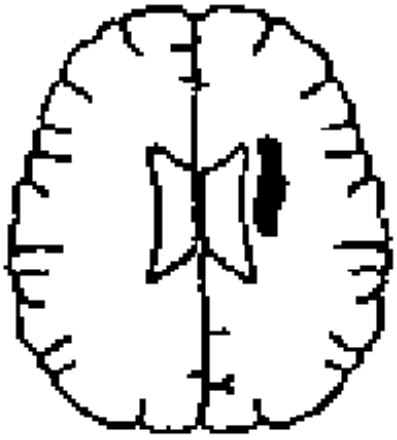
Pattern 4 small embolic infarcts



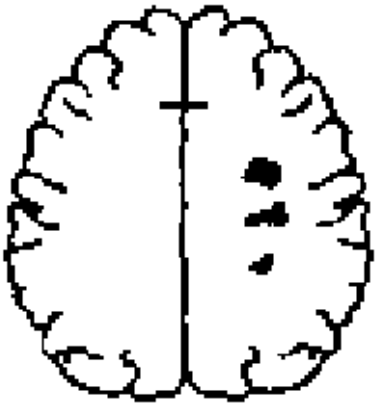
Pattern 5. Anterior Border zone infarct + small posterior border zone infarct



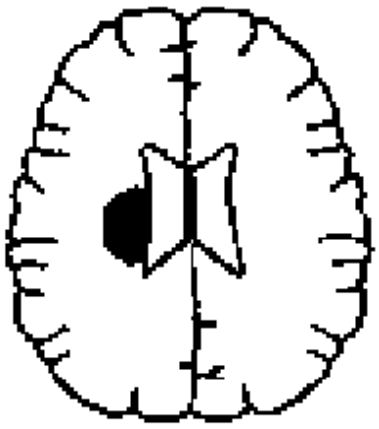
Pattern 5 Posterior border zone infarct



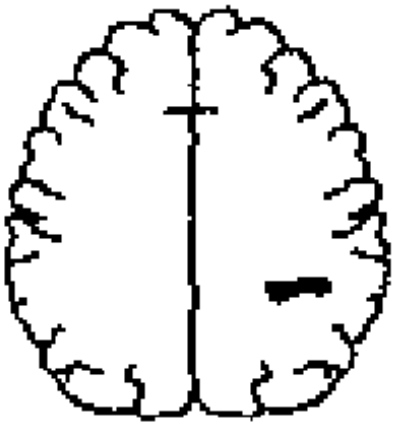
Pattern 5 linear corona radiata infarct



Pattern 5 linear / rosary like centrum semiovale infarct



Pattern 6 discrete corona radiata infarct



Pattern 6 discrete centrum semiovale infarct

RESULTS

TABLE 1

Distribution of Different Patterns of Anterior Circulation Strokes

			Male	Female	Total	Percentage
Pattern1	Partial	Superior	6	3	9	50
		Inferior	5	-	5	
		Unclassified	2	-	2	
	Large	Sup + Inf	2	1	3	18
		Sup + Deep	1	-	1	
		Inf + Deep	2	-	2	
	Total	All 3 divisions	9	-	9	28
	Complete	AC + MC	1	-	1	
Pattern2	Large striatocapsular infarct>2cm.		9	3	12	52
	Lacunar infarcts in striatocapsular region		4	2	6	26
	Discrete corona-radiata infarcts involving striatocapsular region		4	1	5	21
Pattern3	Large fragmentary		10	1	11	
Pattern4	Small multiple		1	1	2	
Pattern5	Cortical anterior border zone		6	1	7	14
	Posterior border zone.		13	4	17	34.6
	Linear corona-radiata		10	3	13	26.5
	Linear centrum semiovale		8	4	12	24.5
Pattern6	Discrete centrum semiovale		3	1	4	
	Discrete corona-radiata		4	4	8	

AC – Anterior cerebral artery
MC – Middle cerebral artery

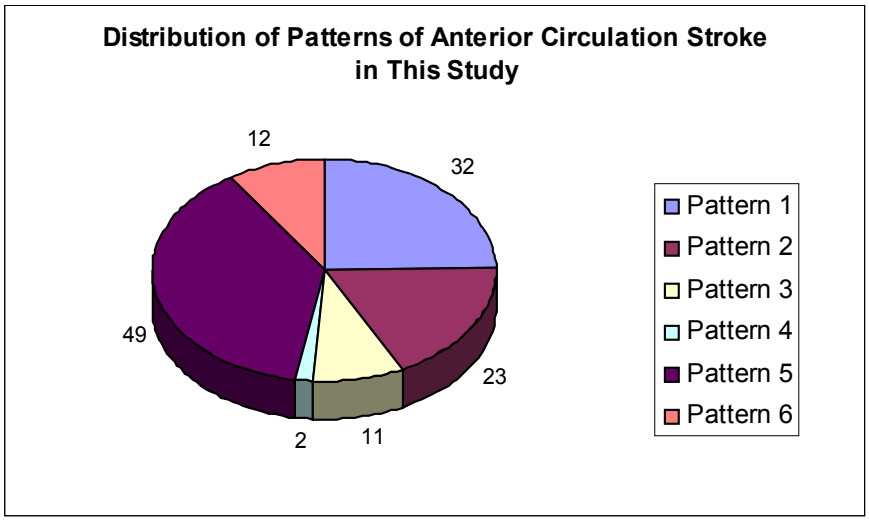

Distribution of Patterns of Anterior Circulation Stroke in This Study
12
32
49
23
2
11
Pattern 1
Pattern 2
Pattern 3
Pattern 4
Pattern 5
Pattern 6

TABLE 2

Cortical Border Zone Infarcts

Anterior Border Zone	Posterior Border Zone	Both
2	13	5

TABLE 3a

Cortical Watershed Associated with Terminal Border Zone Patterns

Anterior border zone alone	1
Posterior border zone alone	1
Both of the above alone	2
Anterior border zone + linear corona-radiata	1
Posterior border zone + linear corona-radiata	2
Both + linear corona-radiata	Nil
Anterior border zone + linear centrum semiovale	Nil
Posterior border zone + linear centrum semiovale	4
Both + linear centrum semiovale	1

TABLE 3b

Cortical Watershed Patterns Associated with Pattern 2

Anterior border zone + large striatocapsular infarct	1
Posterior border zone + large striatocapsular infarct	Nil
Both + large striatocapsular infarct	1
Anterior border zone + lacunar striatocapsular infarct	Nil
Posterior border zone + lacunar striatocapsular infarct	1
Both + lacunar striatocapsular infarct	Nil

TABLE 3c

Cortical Watershed Patterns Associated With Other Patterns

Posterior border zone + discrete centrum semiovale infarct	2
Posterior border zone + superior MCA	1
Both border zone + parietal infarct	1
Posterior border zone + pattern 3	2
Anterior border zone + pattern 3	1

TABLE 4

Association of Striatocapsular and Coronaradiata Infarcts

Total number of striatocapsular infarcts	18
Isolated striatocapsular infarct	9
Lacunar striatocapsular infarct	3
Large striatocapsular + discrete corona-radiata	4
Large striatocapsular + linear corona radiate	2
Lacunar striatocapsular + discrete corona-radiata	1
Lacunar striatocapsular + linear corona-radiata	Nil
Large striatocapsular + discrete centrum semiovale	1
Large striatocapsular + linear centrum semiovale	1
Lacunar striatocapsular + discrete centrum semiovale	1
Lacunar striatocapsular + linear centrum semiovale	Nil

TABLE 5

Association Between Coronaradiata and Centrum Semiovale Infarcts

Linear corona radiate alone	4
Linear centrum semiovale alone	11
Both of the above alone	1
Linear corona radiata + discrete centrum semiovale	2
Discrete corona radiata + discrete centrum semiovale	1
Discrete corona radiata + linear centrum semiovale	1

TABLE 6

Association of Discrete Centrum Semiovale Infarcts

Discrete centrum semiovale + linear corona radiata	1
Discrete centrum semiovale + Posterior border zone infarct	2
Discrete centrum semiovale + multiple small striatocapsular + partial infarct	1

Table 7
Association of Discrete Corona Radiata Infarcts

Discrete corona radiata alone	2
Discrete corona radiata + partial infarct	1
Discrete corona radiata + large striatocapsular infarct	4

Table 8
Association of Linear Centrum Semiovale Infarcts

Linear centrum semiovale infarcts alone	6
With large striatocapsular & discrete corona radiata	1
With lacunar striatocapsular infarct	1
With posterior border zone	1
With partial infarct	3
With lacunar striatocapsular & posterior border zone	1

Table 9
Association of Heart Disease with Infarcts of The Brain

Hypertension	IHD	Rheumatic HD	Atherosclerotic valvular HD
Pattern 1 – 5	Pattern 1 – 3	Pattern 1 – 3	Pattern 5 – 1
Pattern 2 – 1	Pattern 2 – 3	Pattern 2 – 1	Pattern 2,5&6 – 1
Pattern 5 – 3	Pattern 3 – 3	Pattern 5 – 1	
Pattern 5&6 – 1	Pattern 5 – 1		
Pattern 2&6 – 1	Pattern 5&6 – 2		
Pattern 2&5 – 1	Pattern 2&5 – 1		
Pattern 3&5 – 1			
Pattern 2,5&6 – 1			

Table 10
Correlation Between Clinical Profile and Pattern of Strokes

Pattern	Peaks at onset	Progressive	Sensorium normal	Sensorium abnormal
Pattern 1	24	3 (posterior BZ) 2 (multiple pattern) 3 (anterior BZ)	16 (partial) 2 (large)	13 (large) 1 (partial)
Pattern 2	8 (large SC) 2(lacunar) 2 (corona radiata)	4 (large) 4 (lacunar) 3(corona radiata)	20	3 (mixed patterns)
Pattern 3	3	8	5	6
Pattern 4	1	1	1	1
Pattern 5	6 (cortical BZ)	17	5	18
	7 (linear corona radiate & centrum semiovale)	18	17	8
Pattern 6	2	-	2	-

Description of the Tables

Table 1: This describes the various topographical patterns of anterior circulation stroke seen in our study and their distribution by sex. We noted all the six different patterns of anterior circulation stroke in our study which have been described in detail in the section on material and methods.

Large territorial infarction (pattern 1) was observed in 32 patients, in one patient an additional lesion was noted in the anterior cerebral artery territory, suggesting complete carotid artery territory infarction. In our study, partial territorial infarcts comprising either the superior or inferior middle cereberal artery (MCA) form the bulk of large territorial infarctions. We also note that the superior division of the MCA is more involved than the inferior division. 9 cases of total infarcts of the type noted in pattern 1 are seen. Large infarcts sparing at least one division of the MCA and complete carotid artery territory infarctions are less common. We also note that out of the 32 patients with pattern 1 infarcts, only 4 patients are females i.e. only 12.5%. this compares unfavourably with the overall figures in our study in which 22.5% of stroke patients are females. The increased incidence of stroke in males noted in our study could be due to a

referral bias as more males tend to get referred to a tertiary care centre than females, the reason being social.

Pattern 2 subcortical infarctions were noted in 23 patients. Large striatocapsular infarcts were most common with 52% of our patients having them. Small lacunar infarcts in the striatocapsular region and discrete corona-radiata infarcts involving the striatocapsular regions are nearly equal in incidence.

Pattern 3 (large fragmentary) territorial infarcts were noted in 11 patients, of which 10 were males and 1 female.

Pattern 4 (small multiple infarcts in the MCA territory) was the least common; we noted only 2 patients with this pattern in our study.

Pattern 5 (Border zone infarcts) is the most common pattern seen with 49 patients, 37 males and 12 females. We note in our study that cortical and linear subcortical (terminal border zone) patterns are nearly equal in incidence.

Pattern 6 (discrete centrum semiovale and corona-radiata infarcts) are again less common than the other patterns forming about 9% of the total infarcts. We also note that discrete corona-

radiata infarcts are more commonly seen than discrete centrum semiovale infarcts, indicating that involvement of the deep lenticulostriate branches of the MCA which supplies the corona-radiata is more common than that of the superficial perforators of the pial branches of MCA which supplies the centrum semiovale.

In classifying the CT brain images of our patients into these 6 patterns, a problem was encountered with only 2 patients with partial territorial infarcts. These infarcts were in the insular /external capsule regions and therefore classifying them into superior or inferior divisions was difficult. They were included in the category ‘unclassified’ as seen in table 1.

Table 2 describes the number and percentage of patients with anterior/posterior and combined cortical borderzone infarcts. Out of 20 patients with cortical borderzone infarcts, 13 have posterior borderzone alone and 5 have both anterior and posterior borderzone infarcts. Involvement of the anterior borderzone is the least common.

Table 3 describes the association between cortical border zone patterns and other patterns of infarcts.

In table 3a, which describes the association between cortical border zone and linear subcortical (terminal border zone) infarcts, the most common association is between posterior borderzone infarcts and linear (terminal border zone) centrum semiovale infarcts of which 4 such cases are noted. 2 cases of posterior borderzone infarcts being associated with linear (terminal borderzone) corona-radiata infarcts are noted. However anterior cortical border zone infarcts are not associated with linear (terminal) centrum semiovale infarcts at all.

In table 3b, which describes the association between cortical border zone infarcts and striatocapsular infarcts, only 3 cases are noted of which 2 are with large striatocapsular infarcts and one with lacunar striatocapsular infarcts. Considering that we have evaluated 82 patients, this is a very small number.

Table 3C, describes the association between cortical border zone infarcts and other infarcts not mentioned in the earlier two tables. In two cases the cortical borderzone infarcts are associated with discrete centrum semiovale infarcts. These borderzone infarcts are also associated with partial territorial infarcts in 2 cases and form part of pattern 3 i.e. large fragmentary territory infarcts in 3 cases.

Table 4 describes the association of striatocapsular infarcts with linear corona-radiata / centrum semiovale infarcts and discrete corona-radiata / centrum semiovale infarcts. A total of 19 striatocapsular infarcts were noted of which 9 were isolated large striatocapsular infarcts. The most common association was of large striatocapsular infarcts with discrete corona radiate infarcts of which 4 were seen. This association is not the same as discrete corona-radiata infarcts extending into the striatocapsular region of which 5 are already mentioned in Table.1. There are no associations noted between lacunar striatocapsular and linear corona-radiata infarcts. Neither are any noted between lacunar striatocapsular infarcts and discrete centrum semiovale infarcts.

Table 5 describes the association between corona-radiata and centrum semiovale infarcts stressing on differentiation between the linear and discrete infarct types. Isolated cases of linear centrum semiovale infarcts are the most common, 11 of which are noted without association with the other patterns in these two regions. 4 cases of linear corona-radiata infarcts are also noted and 1 case of a linear infarct involving both corona-radiata and centrum semiovale regions are seen. All other

possible associations are seen, though when compared to the total number, these together form only 20% of total.

Table 6 describes the association of discrete centrum semiovale infarcts with other infarcts. This is very uncommon, only 4 cases are noted of which 2 are associated with posterior borderzone infarcts and 1 with the rare pattern 4 described in table 1.

Table 7 describes the association of discrete coronaradiata infarcts with other infarcts. This is more common than discrete centrum semiovale associations, however as noted in table 5, association with large striatocapsular infarcts is the most common, 4 of which are seen. Two cases of isolated corona-radiata infarcts presenting as hemiparesis are also noted.

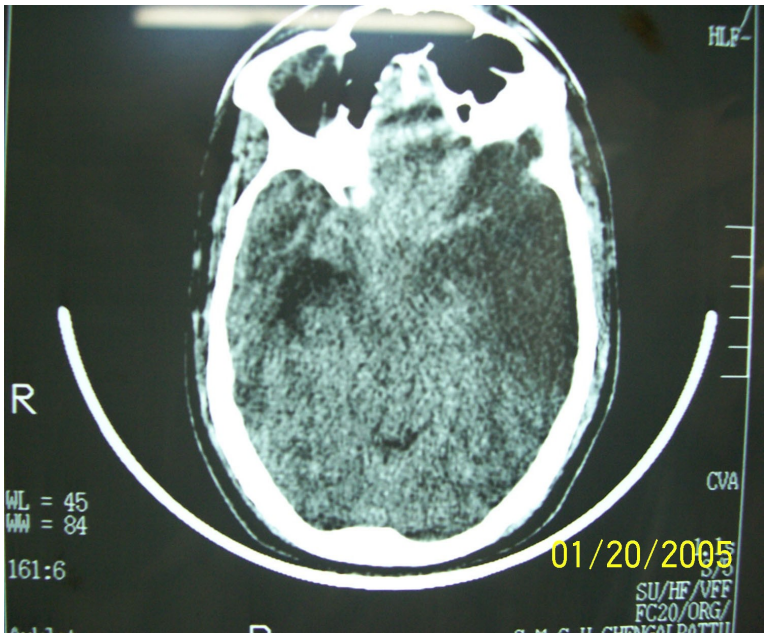
Table 8 describes the association of linear centrum semiovale infarcts with other infarcts. 6 cases of isolated linear centrum semiovale infarcts without any other infarcts presenting as hemiparesis are noted. The linear pattern of centrum semiovale infarct is also associated with territorial, large subcortical striatocapsular and cortical border zone infarts, though the cases are very few as seen in the table.

Table 9 describes the different patterns of infarcts seen in ischemic heart disease, rheumatic heart disease, atherosclerotic valvular heart disease and in patients with ECG and echocardiography evidence of left ventricular hypertrophy probably due to hypertension. In patients with ischemic heart disease, of which there were 15, we note all patterns described in table1 except patients with pattern 4; Patterns 1, 2 and 3 being most common. 14 patients had ECG and echocardiographic evidence of left ventricular hypertrophy. In these patients too, we see all 5 patterns with the exception of pattern 4.

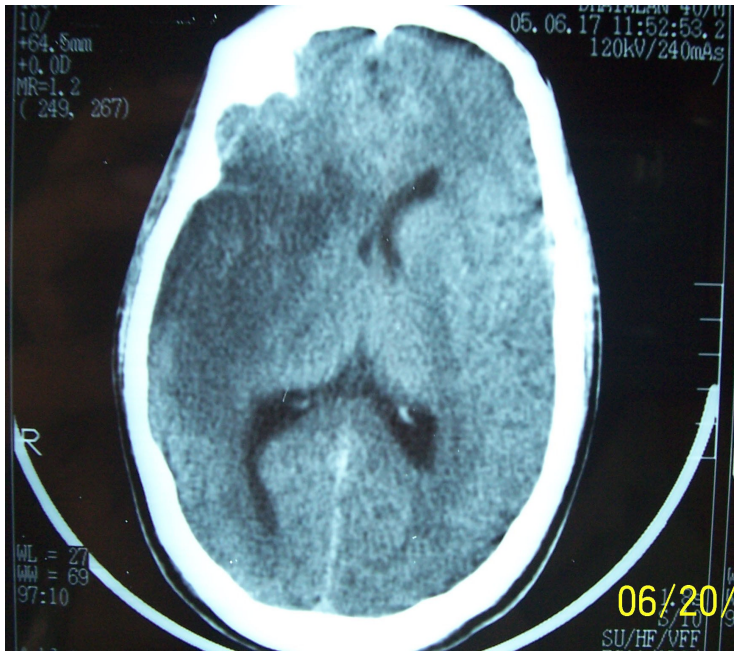
In the 5 patients with rheumatic valvular heart disease, there are 3 patients with pattern 1 infarcts and 1 each of pattern 2 and pattern 5 infarcts. In the 2 patients with athrerosclerotic aortic stenosis, we see pattern 5 alone in one and in combination with pattern 2 and pattern 6 in another. Various other mixed patterns are seen in IHD and left ventriclar hypertrophy, which are shown in the table. Thus we see no specific association between a particular pattern of infarct and heart disease.

Table 10 describes the association between the different patterns of stroke, their clinical evolution and sensorium at onset. In pattern 1, stroke which peaks at onset and then shows

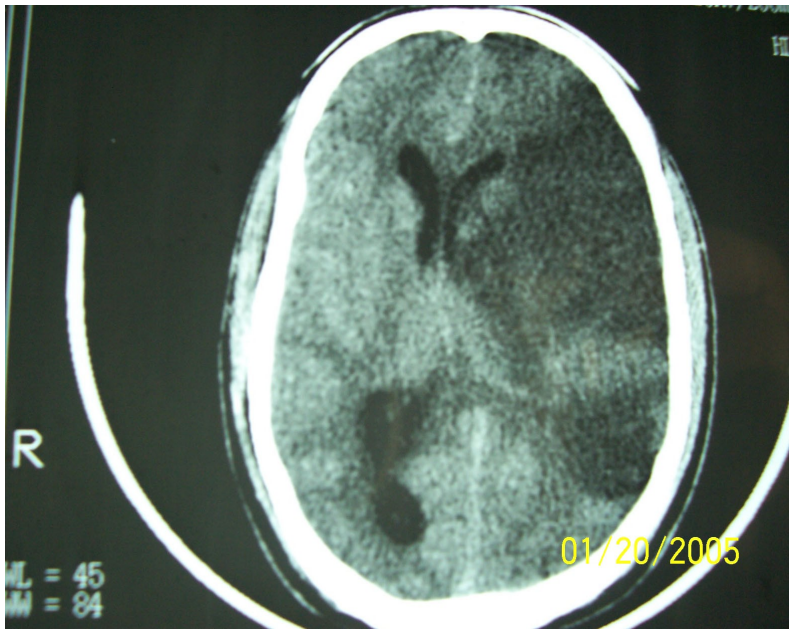
regression are common, 24 such cases being noted out of 32 which is 75% of total pattern 1 strokes. When pattern 1 strokes are progressive, they are usually partial and associated with other patterns as seen in the table. Sensorium at onset is associated with the size of infarct in pattern 1, with large infarcts resulting in altered sensorium and partial infarcts resulting in normal sensorium. In pattern 2, isolated large striatocapsular infarcts are commonly associated with peak at onset and then regression. When two or more areas are involved, progression is noted. Sensorium at onset is normal in pattern 2 except for 3 cases which are associated with other large patterns. In pattern 3, progressive strokes are more common, but sensorium is normal in 5 patients and abnormal in 6 patients. Cortical borderzone infarcts are commonly associated with a progressive course and altered sensorium at onset. Linear subcortical infarcts are commonly associated with a progressive course and normal sensorium. When altered sensorium is seen, they are usually associated with other patterns of infarcts. Discrete isolated corona-radiata infarcts are associated with peak onset and normal sensorium.



(Pattern 1) PARTIAL MCA INFARCT (INFERIOR DIVISION)

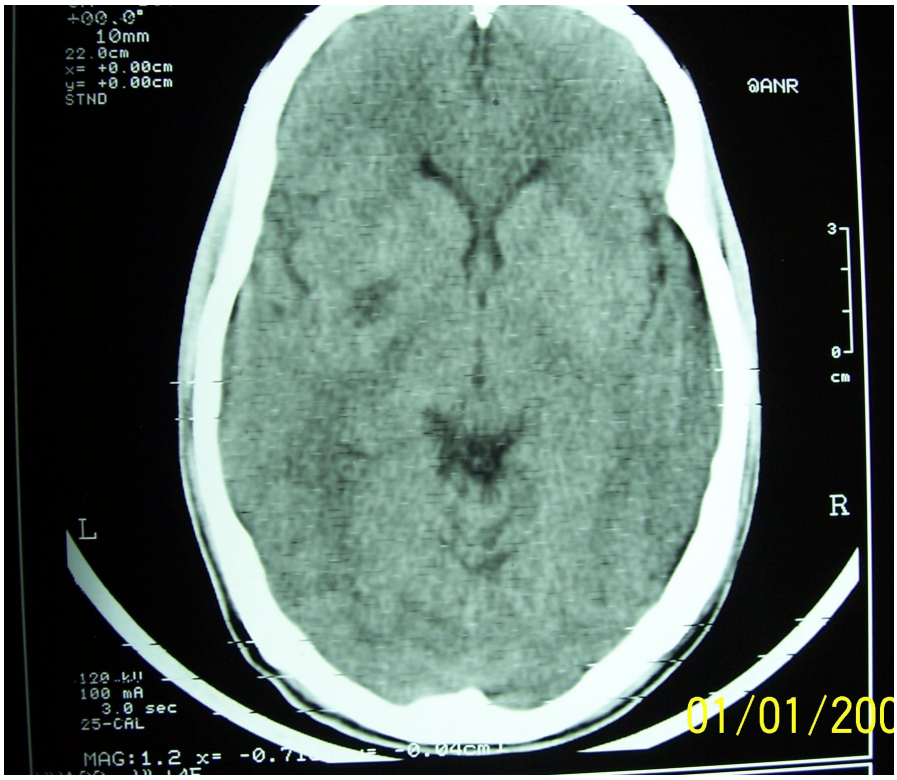


(Pattern 1) PARTIAL MCA INFARCT (SUPERIOR DIVISION)



(Pattern 1) TOTAL MCA INFARCT

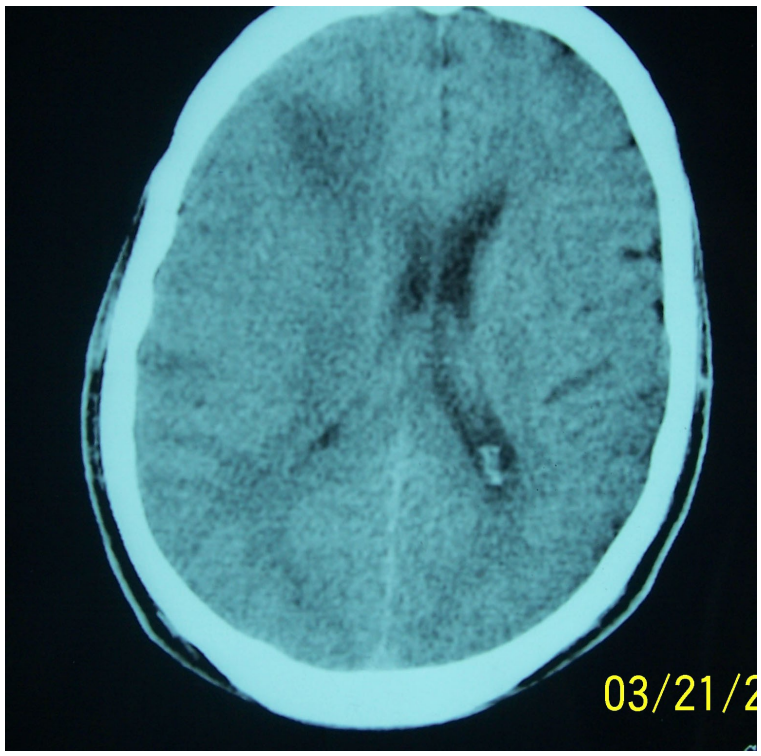
(Pattern 2) LARGE COMMA SHAPED STRIATOCAPSULAR INFARCT



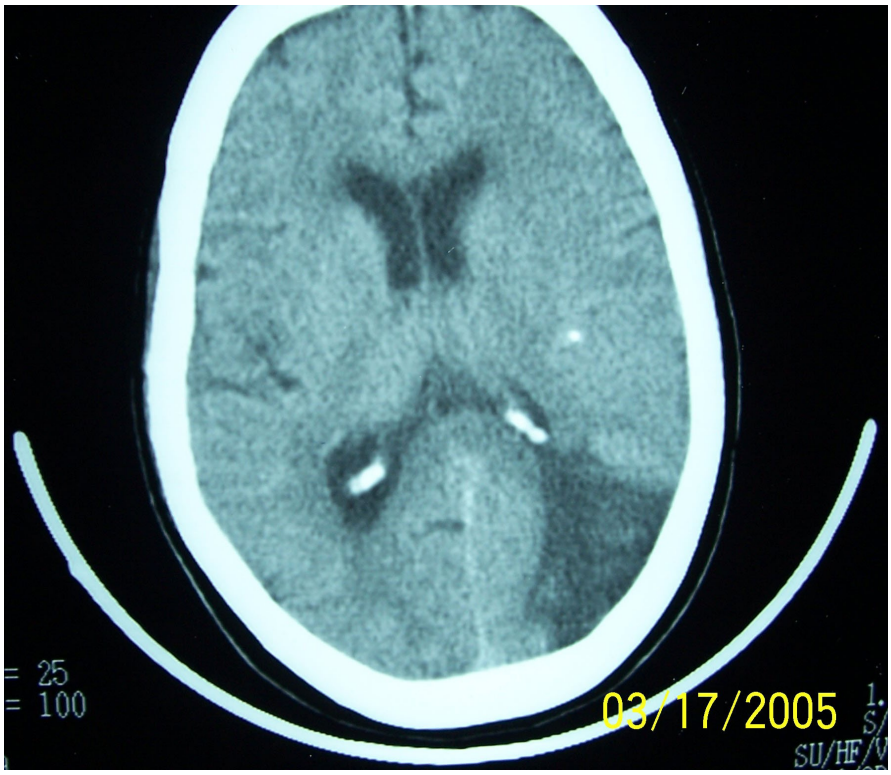
(Pattern 2) MULTIPLE LACUNAR STRIATOCAPSULAR INFARCT

(Pattern 3) LARGE FRAGMENTARY MCA INFARCT

(Pattern 4) SMALL MULTIPLE INFARCTS IN THE MCA TERRITORY

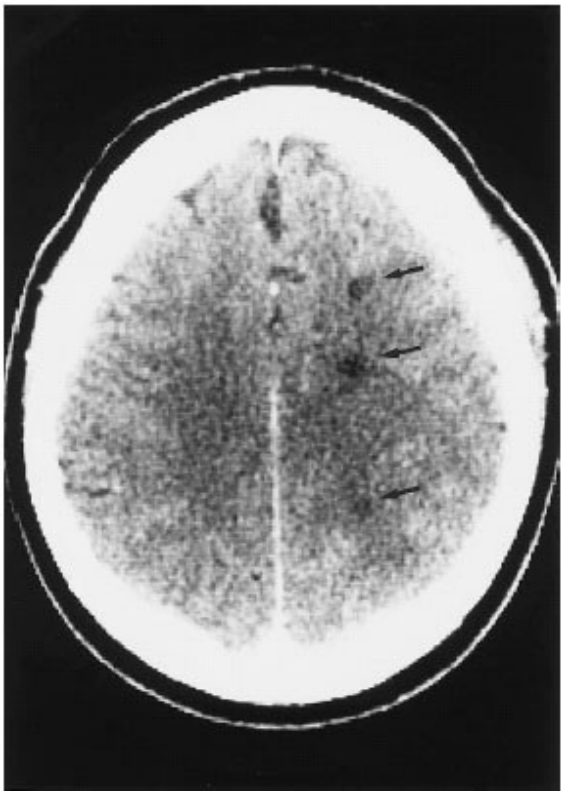


(Pattern 5) ANTERIOR BORDERZONE INFARCT

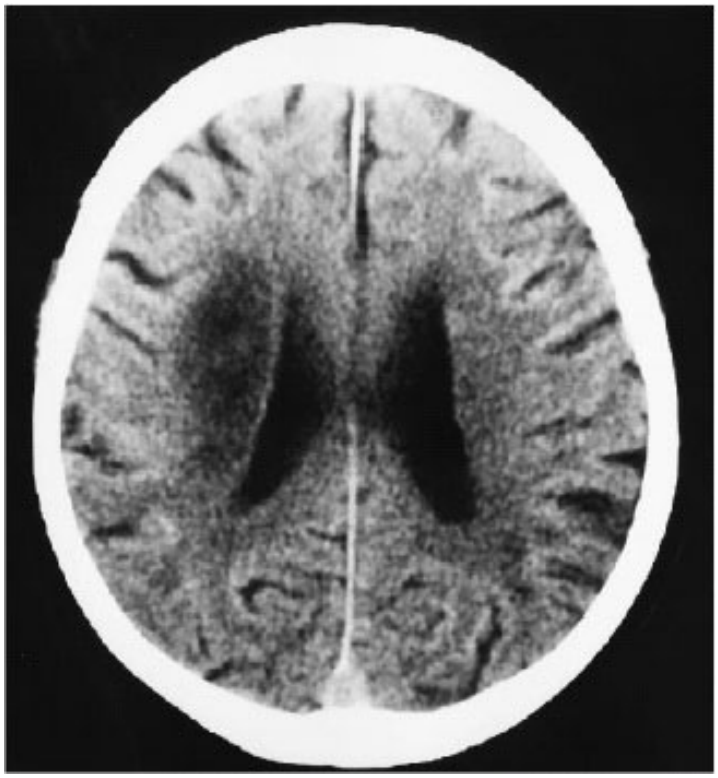


(Pattern 5) POSTERIOR BORDERZONE INFARCT

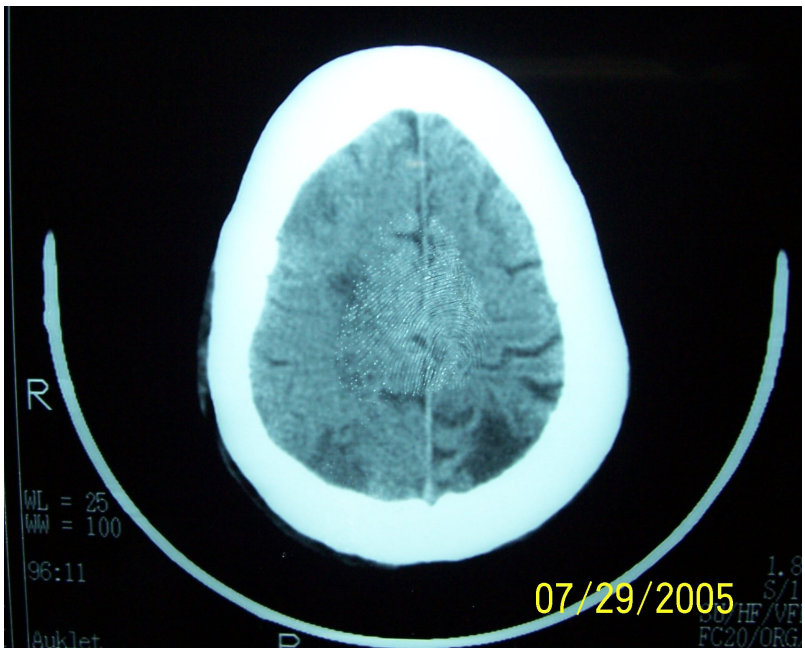
(Pattern 5) LINEAR CORONARADIATA INFARCT



(Pattern 5) LINEAR ROSARY LIKE CENTRUM SEMIOVALE INFARCT



(Pattern 6) LARGE DISCRETE CORONA RADIATA INFARCTS



(Pattern 6) DISCRETE CENTRUM SEMIOVALE INFARCT

DISCUSSION

We had a total of 82 patients with 64 males and 18 females. The increased number of males with stroke have been noted in other studies too,[1,2] but could be due to a referral bias as mentioned earlier.

Topographical analysis of the CT brains of our 82 patients with stroke show that infarcts are seen in all the six different patterns as elucidated by Szabo et al[1] and described in detail in the section on material and methods. Pattern 5, which is borderzone infarction either in the cortical or subcortical regions is by for the most common CT pattern, though it is sometimes associated with patterns 1, 2 and 6. Min et al[2] also find that this is the most common pattern in there topographical analysis of MCA territory infarctions. Cortical and linear subcortical borderzone patterns are equal in incidence in out study unlike Min et al in whose study linear centrum semiovale infarcts are most common. This may be due to an increased incidence of unstable plaques and consequent embolisation to the cortical borderzones in our patients.

Pattern 5 is produced due to haemodynamic compromise of the cerebral circulation due to a cardiac cause or due to occlusive disease of the large cerebral vessels, the internal carotid and middle cerebral artery being by far the most common. CT studies have been used to separate haemodynamic infarctions into two main groups: 1) terminal zone infarctions with a strictly subcortical involvement and location in either the corona-radiata or centrum semiovale[3,4] and 2) watershed or borderzone infarcts with cortical involvement[5,6]. Compared to other types of stroke, such haemodynamically induced infarctions are rare as they occur with insufficient collateralization across the circle of willis and poor leptomeningeal anastomosis[7,8,9]. A percentage of 10% is quoted as the incidence of borderzone infarcts[10]. However, these studies are all done on a Caucasian population.

The large incidence of borderzone infarcts in pattern 5 in our study may be due to racial variations and due to insufficient collateralization across the circle of willis in our patients.

Pattern 1 is the second most common pattern in our study. Partial territorial infarcts involving only one division of the middle cerebral artery is very frequently seen. These pial infarcts are due to embolus from the middle cerebral artery, internal

carotid artery or from the heart. Total infarct of the MCA due to stem occlusions are also common. However, large infarcts with involvement of 2 of the 3 MCA division is less common as the probability of two branches being involved is less than the chance of an embolus obstructing either a division of the MCA or the MCA stem as a whole. Complete AC + MC infarcts seen in one of our patients may be associated with distal ICA occlusion, though we do not have Doppler and angiographic studies to substantiate it. However ICA stenotic or occlusive disease can also produce all pattern 1 type infarcts, pattern 2 infarcts and cortical border zone and linear centrum semiovale infarcts as in pattern5[12].MCA stenotic and occlusive disease can also produce pattern 1 infarcts excluding complete AC + MC infarcts. It can also produce pattern 2 and pattern 5 cortical borderzone infarcts and linear corona-radiata infarcts. Thus from pattern 1 topography, it is difficult to tell which artery is involved unless both AC and MC are involved. But pattern 1 infarcts indicate that there is definite involvement of the large arteries of the brain and thus is an ideal starting point to plan neurointervention if feasible.

Pattern 3 infarcts are also noted in this study. They represent large vessel embolic occlusions with subsequent fragmentation of the embolus[1,13]. This is different from pattern 4 where several disseminated small lesions are sprinkled in random fashion in the distal territory of the MCA, involving mainly cortical but also subcortical regions. The possible cause of this pattern may be a fragmented embolus are multiple microemboli in small vessels[14], we noted only 2 patients with this pattern in our study. This is similar to other studies[15], which too show that this pattern is very uncommon.

Pattern 6 infarcts are discrete centrum semiovale and corona-radiata infarcts. They are not lacunar infarcts as their size is >1.5cm as suggested by Bogouslavsky[3] and thus are due to atherothrombotic or cardioembolic cause. This pattern is also noted in our study.

Cortical borderzone infarcts involve the anterior region between the territories of the ACA and MCA and the posterior region between the territories of the ACA, MCA and PCA. As the cortical regions have a rich pial collateral blood supply, anterior borderzone infarcts theoretically can only occur with either internal carotid artery disease or more proximal pathology.

Similarly, posterior borderzone infarcts can only occur when the disease is in the arch of aorta or in the heart. This should also be associated with insufficient cross collateralization in the circle of willis. The only other possibility when such borderzone infarcts occur is when both MCA and ACA are stenosed in anterior borderzone infarcts and the MCA, ACA and posterior cerebral arteries are involved in posterior borderzone infarcts. This is statistically less likely, though possible. In our study, posterior borderzone infarct is most common which would mean disease of the arch of aorta or the heart, but these should ideally be associated with anterior borderzone infarcts and be bilateral too. Some authors[16,17,18,19] have suggested that embolisation may play an important role in pathogenesis of these infarcts. Pollanen et al[17] reported their autopsy cases of the anterior borderzone infarctions caused by thromboemboli. In their autopsy series, Masuda et al[20] reported that atheroembolism in the brain frequently caused anterior and posterior borderzone infarcts by occluding the terminal cortical branches. Thus, emboli of a certain small size might occlude terminal portions of the cortical branches and cause a so called watershed infarction. This might explain why isolated posterior borderzone infarcts are noted in

our study and noted so commonly. It might also explain why some of these posterior borderzone infarcts in our study are fragmentary in nature, breaking of the embolus being the reason. Caplan et al[21] emphasized the interaction of hypoperfusion and embolization, i.e decreased perfusion reduces the washout and clearance of emboli that enters the vascular bed of hypoperfused regions.

Since the cortical borderzone infarcts can be due to haemodynamic and embolic cause, we tried to look into the associated stroke patterns with these infarcts in Table 3. Linear centrum semiovale[12] and linear corona-radiata infarcts[3,4] in a rosary pattern or confluent and large > 1.5cm are due to haemodynamic failure. It was noted that the most common association was between posterior borderzone infarcts and linear centrum semiovale infarcts. Krapf[12] suggests that the linear centrum semiovale infarcts are due to stenosis of the internal carotid and consequent haemodynamic failure. Hence, their association with posterior borderzone infarcts indicate the following possibilities borderzone infracts indicate the following possibilities 1) embolic aetiology for the borderzone infarcts 2) associated posterior circulation, arch or cardiac aetiology.

However, as earlier mentioned, the absence of anterior borderzone infarcts indicates that the likely cause is embolic from the internal carotid rather than haemodynamic failure. Similarly, the association of posterior borderzone infarcts with linear / large corona-radiata infarcts which are due to MCA stenotic disease and consequent haemodynamic failure would also indicate that the borderzone infarct is embolic in origin. The absence of anterior borderzone infarcts with linear centrum semiovale infarcts is also against the internal carotid artery haemodynamic failure theory of anterior borderzone infarcts. Thus, the study suggests as do the other studies already mentioned, that cortical borderzone infarcts can be embolic in origin too.

Large striatocapsular infarcts > 2 cm in size with a comma like, lens like or triangular configuration usually occurs when several lenticulostriate arteries are simultaneously exposed to ischemia, and there is sufficient cortical blood flow attributable to rich leptomeningeal collaterals[4,22,23]. Occlusion of the orifice of the lenticulstriate arteries at the level of M1 segment of MCA, either atherothrombotic permanent occlusion or embolic transient occlusion, has been supposed to be a major cause of

striatocapsular infarcts. The very small number of large striatocapsular infarcts associated with cortical borderzone infarcts indicate that emboli from these MCA plaques are rare. Thus it may be that the large striatocapsular infarcts are due to sudden occlusive disease of the MCA, either atherothrombotic or embolic without subsequent fragmentation of the clot unlike the slow stenotic pathology seen in linear corona-radiata infarcts. This assumption is also substantiated by our findings in table 10 where large striatocapsular infarcts peaks immediately clinically.

As seen in table 3C, cortical borderzone infarcts are also associated with discrete centrum semiovale infarcts. Phil Hyu Lee et al[15] suggest that discrete centrum semiovale infarcts are due to superficial perforating artery involvement. These are the terminal medullary penetrating arteries of the superficial MCA branches and are involved due to emboli from the heart or internal carotid artery[24]. It is difficult to distinguish them from lacunar strokes due to primary disease in these vessels itself. The only clue would be the multiplicity and bilaterality of findings if it were due to small vessel disease. As we have excluded isolated single centurm semiovale infarcts of size < 1.5cm and the other discrete centiam semiovale infarcts in our study are associated

with haemodynamic failure / embolic pattern, we can safely assume that infarcts in our study too are due to embolic cause as suggested by Yonemara et al[24]. The association of cortical borderzone infarcts with partial territorial infarcts and pattern 3 infarcts is further evidence of the combined haemodynamic / embolic theory of their origin.

In table 4, of the 18 striatocapsular infarcts 9 are isolated large infarcts. This indicates that MCA occlusion with very good leptomeningeal collateralization is frequently seen in our set up. Large striatocapsular infarcts are commonly associated with discrete corona-radiata infarcts. For the pattern to occur, either the corona-radiata infarcts are an extension of the striatocapsular infarct or that area of the corona-radiata is supplied by the superficial penetrating artery and the infarct is due to its occlusion. We assume it is the former, as also suggested by Nakano et al[4], though the latter possibility cannot be ruled out. However, by definition[15], superficial perforator infarct's innermost limit is the corona-radiata at the deep perforator level. An isolated infarct with predominant involvement of the corona-radiata without extension into the centrum semiovale is more likely to be due to extension of a striatocapsular infarct.

In table 5, the most common pattern noted is isolated cases of linear centrum semiovale infarcts. This pattern of infarcts is commonly due to haemodynamic failure from an internal carotid artery stenotic disease[3,12]. 4 cases of linear corona-radiata infarcts are also noted. Zulch's work[25] has shown that periventricular white matter of the corona-radiata is the watershed zone between the deep and superficial territories of the MCA. Linear / confluent infarcts in this region are due to haemodynamic failure due to MCA disease. The association of linear infarcts in both centrum semiovale and corona-radiata in one case could be due to tandem lesions in the IC and MC. The association of linear corona-radiata with discrete centrum semiovale infarcts could be due to emboli in the centrum semiovale (superficial perforator) territory from an atherothrombotic middle cerebral artery as also the association between discrete corona-radiata and discrete centrum semiovale infarcts. The association of discrete corona-radiata and linear centrum semiovale infarcts could be due to distal emboli from an internal carotid atheroembolic disease.

Discrete centrum semiovale infarcts are due to involvement of the superficial perforators[15,24] as already mentioned and are

embolic in origin. In table 6, we tried to look into its association with other patterns of stroke. It is associated with posterior borderzone infarcts in 2 cases which could be postulated as embolic. Its association with the rare pattern 4 in one case, which is essentially embolic in nature, is consistent with embolic origin of superficial perforator infarcts.

Linear centrum semiovale infarcts[12] as already mentioned are haemodynamic in origin and mainly due to IC disease. In table 8, 6 cases of isolated centrum semiovale infarcts are described. Linear centrum semiovale infarcts are also associated with partial MCA infarcts, borderzone infarcts and striatocapsular infarcts. All these patterns can happen with ICA disease, though linear CSO infarcts are never seen with MCA disease[12].

From the findings in table 9, we can conclude that there is no pattern of stroke which is specific for heart disease. Stroke due to heart disease could be due to embolus or haemodynamic failure and hence can result in all patterns of stroke and various combinations too. Hence, it is essential to evaluate the heart in every case of stroke.

Episodic, fluctuating or progressive weakness and shaking of lower limb in the absence of amaurosis fugax is usually indicative of haemodynamic failure[26,27]. Syncope at onset, often precipitated by postural change is also haemodynamic in origin. Sudden onset of symptoms with subsequent static or regressive course is seen with embolic or atherothrombotic sudden occlusive lesions[28]. In table 10, we see that pattern 1 and isolated large striatocapsular pattern 2 infarcts are commonly associated with sudden onset (peaks at onset) of neurological symptoms. This is consistent with their pathophysiology being due to embolus or sudden atherothrombotic occlusion of the MCA. Pattern 3 being progressive in nature is against our assumption of them being due to emboli in large vessels and their subsequent lysis. Therefore, another explanation could be that these strokes are due to large vessel atherothrombotic disease with unstable plaques which floridly send emboli distally. Pattern 5 is generally progressive, they being due to an ongoing haemodynamic / embolic aetiology in the large vessels.

Sensorium at onset is dependent on the size of brain tissue involved and not on any pattern of CT Brain involvement

according to table 10, with large Pattern 1 infarcts almost always having altered sensorium at the onset of stroke.

CONCLUSION

82 patients, both male and female were included in the study satisfying the defined inclusion and exclusion criteria. Analysis of their CT brains resulted in the following conclusions.

1. Infarcts in the anterior circulation fit into 6 different patterns namely
    a. Large territorial or cortical infarcts
    b. Large / discrete subcortical infarcts involving he striatocapsular and corona radiata region.
    c. Large fragmentary infarcts in the MCA/ACA territory.
    d. Small fragmentary infarcts in the MCA/ACA territory
    e. Cortical borderzone infarcts and subcortical borderzone i.e linear corona radiata and linear centrum semiovale infarcts.
    f. Discrete corona-radiata and discrete centrum semiovale infarcts.

Among the 6, pattern 5 or border zone infarcts are most common.

2. 2 or more of these patterns can co-exist, though there are no definite combinations. Combinations vary depending on the proposed pathophysiology of stroke.
3. Cortical borderzone infarcts usually thought to be due to haemodynamic failure can be embolic in origin too.
4. Heart disease can result in infarcts in any pattern or their combinations thereof.
5. Pattern1 cortical infarcts and pattern 2 large striatocapsular infarcts show sudden peak clinically with a subsequent static or regressive course.
6. Progressive strokes are usually associated with combinations of different patterns in CT Brain
7. Sensorium at onset is associated more with the size of the infarct than with a pattern of infarct.

It is difficult to correctly guess the artery involved in anterior circulation strokes from the pattern of CT Brain involvement and the clinical course, though certain patterns do indicate possible aetiologies.

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NAME IP NO:
AGE MIN NO:
SEX SR NO:
DOA D0D:

I.

A. TIME OF ONSET OF STROKE.
:

B. TIME OF ONSET TO TIME OF MEDICAL HELP :

C. TIME OF ONSET TO TIME OF HOSPITALISATION :

D. TIME OF ONSET TO TIME OF REFERAL TO NEUROLOGIST/TERTIARY CARE:

E. TIME OF ONSET TO TIME FOR CT SCAN REPORT :

F. TIME OF ONSET TO TIME FOR TREATMENT
MEDICAL :
PHYSIOTHERAPY :
INTENSIVE CARE :
SURGICAL :

II.AWARENESS OF STROKE PATIENT CLOSE RELATIVE

A. HAVE YOU HEARD OF STROKE?

B. WHAT IS STROKE?
(MARK CORRECT/WRONG)

C.WHAT DISABILITY CAN IT CAUSE?
(MARK CORRECT/WRONG)

D.WHAT IS THE CHANCE OF RECOVERY?
(MARK CORRECT/WRONG)

E.WHAT ARE THE RISK FACTORS?
(MARKCORRECT/WRONG)

F.DOES IT REQUIRE IMMEDIATE MEDICAL ATTENTION?

G.HOW DOES IT COMPARE WITH HEART ATTACK?
SAME/MORE/LESS
PATIENT
RELATIVE

H.IS IT LIFE THREATENING?

I. WHAT ARE THE TYPES OF STROKE?

J. HOW WERE YOU AWARE OF STROKE?
NEWS PAPER
FRIENDS TV/RADIO OTHERS (INDICATE), DOCTOR
K. CAN IT BE PREVENTED? HOW?

III. STROKE RECURRENCE.

	Ist	IInd	III rd	IV th	Vth	MORE
A.NO. OF EPISODES (IN YEARS) TYPE						

B.WHETHER ON ANTIPLATELETS
ANTICOAGULANTS
STATINS

C.WHETHER HYPERTENSIVE UNDER CONTROL?
DIABETIC UNDER CONTROL?

D.WHETHER CONTINUED TO SMOKE, DRINK OR STOPPED?

E. WHAT ARE THE OTHER RISK FACTORS IDENTIFIED?

F.WHETHER ANY LIFE STYLE MODIFICATION ATTEMPTED?WHAT?

IV STROKE HISTORY

A) TIME OF ONSET –

B) WHAT WAS THE PATIENT DOING AT ONSET ?

EXERTION
REST
SLEEP
MEALS
SEX
IN HOSPITAL PROCEDURE
IN BATHROOM
DOING HOUSE WORK
OTHERS (INDICATE)

C) WHAT WAS HIS MOOD BEFORE / DURING STROKE ?
HAPPY
SAD
NORMAL
NOTHING TO MENTION

D)WHAT WERE HIS PRECEDING SYMPTOMS ?

HEADACHE	SENSORY
(INDICATE TYPE)	MOTOR
VISUAL COMPLAINTS	VERTIGINOUS
(INDICATE TYPE)	ATAXIC
ANXIETY	DIZZY / GIDDY
NEUROPSYCHIATRIC	OTHERS

E)WHAT WAS THE PROGRESSION OF STROKE TILL INTERVIEW ?

PROGRESSIVE
STUTTERING PROGRESSIVE
PEAKS IMMEDIATELY / STATIC
REGRESSION
REGRESSION / PROGRESSION

[GIVE A BRIEF HISTORY]

F) HOW DID THE ASSOCIATED SYMPTOMS APPEAR IN CHRONOLOGY ?

I ST SYMPTOM
II ND SYMPTOM
III RD SYMPTOM
IVTH SYMPTOM
V TH SYMPOM
OTHERS

G) WHAT ARE THE ASSOCIATED NON NEUROLOGICAL SYMPTOMS

CHEST PAIN FEAR ABD PAIN URINARY COMPLAINT
SWEATING OTHERS (INDICATE)

H) SENSORIUM AT ON-SET - LOC
WHEN
(DESCRIBE IN 2 LINES)

I) WAS SPEECH DISTURBANCE NOTED AT TIME OF STROKE ?

WHEN ?
WHAT TYPE-EXPRESSIVE
SENSORY
GLOBAL
DYSARTHRIA
MUTISM

J) SLEEP - DID HE SLEEP WELL DAY BEFORE STROKE ?
HOW LONG DOES HE SLEEP NORMALLY?
DOES HE WAKE UP EARLY OR SLEEP LATE?
HAS SLEEP BEEN AFFECTED AFTER STROKE?
HOW?

K) HISTORY OF TIAS WHEN ACTIVITY AT TIME OF TIA

MOTOR WEAKNESS
DYSPHAGIA
IMBALANCE
SPEECH
SENSORY
VISUAL
VESTIBULAR
MEMORY
BEHAVIOURAL
OTHERS

CHANGE OF POSTURE
NECK TURNING
EXPOSURE TO BRIGHT LIGHT
SUNLIGHT
HOT BATH
MEALS
EXERCISE
SEX
BATHROOM
AFTER TAKING DRUGS
eg hypotensive
PALPITATIONS

HAVE WE RULED OUT D.D.s OF TIA ? (KEEP CHECK LIST)

MIGRAINE AURA SEIZURES TGA LABYRINTHINE DISORDERS

METABOLIC HYPERVENTILATION / PANIC MS

SYNCOPE DROP ATTACKS TUMORS

L) RISK FACTORS. SINCE WHEN ON TREATMENT/NO CONTROLLED
(M/NM)

HTN
CAD
VALVULAR
AF
DM
SMOKING
TOBACCO
OTHER ADDICTIONS
OLD AGE (N/M)
SEX
OCPs
PREGNANCY
POST PARTUM
BLEEDING DIATHESIS
CLAUDICATION
SHOCK
TRAUMA
MALIGNANCIES
IMMOBILITY
SICKLE CELL
THROMBOPHILIAS
DRUG ABUSE /OTHERS

TYPE BRAND NUMBER DURATION

CIGARETTES

ALCOHOL

TOBACCO

M) WHAT ARE ASSOCIATED DISEASES AND SYSTEMS INVOLVED?

CVS

P/A

R/S

MUSCULOSKELETAL

OTHER CNS SYSTEMS

PNS

CORD

OTHERS

MIGRAINE

OTHER HEADACHES

SEIZURES

MOOD DISORDERS

OTHERS

N) DIET

VEG NONVEG EGG+VEG

TYPE OF OIL USED

ANY FOOD FADS

O) TREATMENT/DRUG HISTORY?

WHAT DRUGS?

IS HE REGULAR/COMPLIANT?

ANY SIDE EFFECTS?

P) WHAT IS THE NATURE OF WORK?

SEDENTARY MODERATE HEAVY EXERTION

Q) FAMILY HISTORY

	1st degree	2nd degree	OTHERS
STROKE			
TIA			
CAD			
DM			
HTN			
SMOKING			
ALCOHOL			
OTHERS			
SUDDEN DEATH			

V : CLINICAL EXAMINATION

A) SKIN XANTHOMAS ARCUS

TAO

ISCHEMIA

B) BRUIT- CAROTIDS ORBITAL SUP.TEMPORAL OTHERS

PULSES-	R	L
CAROTIDS		
SUBCLAVIAN		
BRACHIAL		
RADIAL		
ULNAR		
FEMORAL		
POPLITEAL		
DORSALIS PEDIS		
FRONTAL ARTERY SIGN		

	B.P	PULSE
SUPINE		
SITTING		

E) BMI (WT/HT2)
WAIST/HIP RATIO

F) OCULAR FUNDUS-
PAPILLOEDEMA
HTN/DM
RETINOPATHY
EMBOLI
OTHERS

G) CONSCIOUSNESS- NORMAL
DROWSY
STUPOR
COMA

H) LOBAR FUNCTIONS

FRONTAL : Apathy
Agitation
Judgement
Verbal flexibility
Motor persistence
Gaze deviation

Parietal : Cortical sensation
Apraxia
Finger body gnosis
Neglect

Occipital : Vision
Face/ object recognition

Temporal : Memory verbal
Visual

I) CRANIAL NERVES

J) MOTOR SYSTEM

TONE: +

POWER:	Face		UL		LL	
Weakness	R	L	R	L	R	L

Reflexes Corneal
Abd
Plantar

DTR	B	T	S	K	A
R					
L					

K) SENSORY	UL		LL	
	R	L	R	L
Touch				
Pain				

L: Cerebellar Signs
M: EP Signs
N:Bladder Symptoms – Urgency
Frequency
Retention
Whether catheterized?
When?
Bowel Symptoms
O: Neck Rigidity
Kernig's

P) OTHER SYSTEMS

CVs MUSCULOSKELETAL

R/S

P/A

VI) INVESTIGATIONS

A) Hb	RBS	MCV
Tc	FBS	Lipid Profile
DC	PP BS	T. Chol
ESR	B1 Urea	VLDL
Thin PS	S Creat	HDL TGL others

B) ECG - O/A
O/D
D) ECHOCARDIOGRAPHY

D) C X R PA VIEW
E)IMAGING

CT BRAIN TIME WHEN TAKEN
HOW MANY HRS AFTER STROKE

HOW MANY HRS AFTER ADMISSION

CAUSE OF DELAY – MONETARY
PROCEDURAL
OTHERS

WHETHER INFARCT
HEMORRHAGE
HEMORHAGIC INFARCT
SAH
OTHERS (INDICATE)

IF INFARCT – CORTICAL
SUBCORTICAL
LARGE
LACUNAR

AGE OF INFARCT

OTHER ASSOCIATED INFARCT & SITE

IF HEMORRHAGIC TRANSFORMATION
WHETHER GYRAL
SUBCORTICAL
GENERALISED

VENOUS THROMBUS SEEN :
(DESCRIBE)
MIDLINE SHIFT

OEDEMA MILD MODERATE SEVERE

OTHER CT SIGNS - DENSE MCA
INSULAR RIBBON
EFFACED SULCI
GREY / WHITE DIFF
CORD SIGN
EMPTY DELTA
HYDROCEPHALUS

IF HEMORRAGE – SITE
(Describe extent) Size < 10mm 4Cm > 4cm
Surrounding Oedema
(as %of hemorrhages)
cortical / Sub cortical
Intraventricular Extension
Suboranchond extenion
Cisterne effoaced
Hydrocephale

If SAH Area / Vessel

Cisterne involved
Hydrophela
Thrombosed Aneurysm

CT CONTRAST

(DESCRIBE CHANGE)

Rpt CT BRAIN
Describe Changes)

MRI BRAIN

Areas involved

MRA - Whether Vessels Involved are seen

4 Vessels Doppler - CAROTIDS FLOW DIANETER PLAQUE

R CCA
L CCA
R ICA
L ICA
R VERTEBRAL
L VERTEBRAL

4VESSEL ANGIO DESCRIPTION

MASTER CHART

S. No.	Age/ Sex	Type of Prog	Senso	ECG	Echo	CORTICAL / TERRITORIAL INFARCT				SC infarct	DCSO	LCSO	DCR	LCR	FSC	PSC	Patt 3	Patt 4	Leuko araiosis	ABZ	PBZ
						Partial	Large	Total	Complete												
1.	28/M	P	AB	N	N			+													
2.	63/M	P	AB	LVH	LVH			+													
3.	38/M	Pr	N	N	N												+				
4.	65/F	Pr	N	N	N												+				
5.	40/M	P	N	LVH	LVH					L											
6.	22/M	Pr	N	N	N					L				+						+	
7.	69/M	Pr	N	N	N						+										+
8.	60/M	Pr	N	IHD	IHD						+			+					+		
9.	49/M	Pr	N	N	N					Lac					+						
10.	50/M	Pr	N	N	N							+									
11.	45/F	P	N	IHD	IHD					L					+						
12.	80/F	Pr	AB	N	N						+						+				+
13.	63/M	P	AB	LVH	LVH		+														
14.	36/M	P	AB	N	N			+													
15.	46/M	P	N	N	N												+				+
16.	55/M	Pr	AB	LVH	LVH									+							+
17.	75/M	Pr	N	LVH	LVH					L				+	+	+			+		
18.	55/M	Pr	N	N	N				+								+			+	
19.	65/F	Pr	AB	IHD	IHD															+	+
20.	65/M	P	AB	N	N												+				
21.	55/F	Pr	AB	N	AS					L		+	+								
22.	60/F	Pr	AB	LVH	MS									+					+		
23.	72/M	P	AB	N	DD		+														
24.	60/M	Pr	N	N	N					Lac		+									
25.	48/M	Pr	AB	IHD	IHD	+															+
26.	82/F	Pr	AB	N	N													+		+	+
27.	54/M	Pr	N	N	N					Lac	+	+									
28.	50/M	P	AB	N	N							+									+
29.	35/M	P	AB	IHD	IHD			+													
30.	65/M	Pr	N	AF	N			+									+				
31.	56/M	Pr	N	IHD	IHD			+									+				+
32.	20/M	P	AB	N	N					L			+								
33.	38/M	Pr	N	N	N	+															
34.	50/M	P	AB	IHD	IHD												+				
35.	36/M	Pr	N	AF	CLOT	+															

36.	54/M	P	AB	N	LVH					Lac			+	+					+		

S. No.	Age/ Sex	Type of Prog	Senso	ECG	Echo	CORTICAL / TERRITORIAL INFARCT				SC infarct	DCSO	LCSO	DCR	LCR	FSC	PSC	Patt 3	Patt 4	Leuko araiosis	ABZ	PBZ
						Partial	Large	Total	Complete												
37.	60/M	P	N	N	N	+															
38.	46/M	P	AB	CHB	LVH		+														
39.	53/M	P	N	IHD	IHD								+								
40.	50/M	Pr	N	LVH	LVH												+				+
41.	57/M	P	N	N	N	+						+			+	+					
42.	60/F	Pr	N	LVH	LVH	+				L											
43.	40/M	P	N	N	N			+													
44.	50/M	P	N	N	N	+								+							
45.	56/F	P	N	N	N								+								
46.	49/F	P	AB	LAE	MS		+														
47.	28/M	P	N	N	N												+				
48.	60/M	P	AB	LVH	N									+					+		
49.	64/M	Pr	AB	IHD	IHD							+									
50.	63/F	P	N	N	N	+						+									
51.	40/F	P	AB	N	MS					L			+								
52.	70/F	P	N	N	DCM					L											
53.	62/M	P	AB	N	N			+													
54.	20/M	P	N	N	N		+														
55.	50/M	P	AB	N	N		+														
56.	45/M	P	AB	N	N												+				
57.	70/M	P	N	N	N	+															
58.	47/M	P	N	N	LVH							+		+					+		+
59.	60/M	Pr	N	LVH	LVH									+		+			+		
60.	60/F	P	N	LVH	N								+	+							
61.	38/M	P	N	N	N	+															
62.	60/M	P	N	N	N					L											
63.	55/M	Pr	N	N	N	+						+								+	+
64.	60/M	Pr	N	IHD	IHD					Lac		+									+
65.	37/M	Pr	N	N	N	+															
66.	83/F	P	N	N	N							+									
67.	50/M	P	N	N	N									+							
68.	58/M	P	N	IHD	IHD													+			
69.	56/M	P	AB	IHD	IHD			+													
70.	37/M	P	N	N	N	+				L			+								

71.	58/M	Pr	AB	IHD	N	+														+
72.	54/M	Pr	N	N	N							+				+				

S. No.	Age/ Sex	Type of Prog	Senso	ECG	Echo	CORTICAL / TERRITORIAL INFARCT				SC infarct	DCSO	LCSO	DCR	LCR	FSC	PSC	Patt 3	Patt 4	Leuko araiosis	ABZ	PBZ
						Partial	Large	Total	Complete												
73.	75/M	P	N	N	N					L											
74.	24/M	P	N	N	N					L											
75.	65/M	Pr	N	N	LVH	+															
76.	52/M	Pr	AB	N	N				+												
77.	45/F	P	AB	LVH	N							+									
78.	55/F	P	AB	N	MS	+															
79.	52/M	P	N	LVH	N												+				
80.	67/M	P	AB	LVH	AS															+	+
81.	75/F	Pr	N	LVH	N					Lac											+
82.	45/M	P	N	N	N									+							

M – Male
F – Female
P – Peaks at onset
Pr – Progressive
AB – Abnormal
N – Normal
LVH – Left Ventricular Hypertrophy
IHD – Ischemic Heart Disease
AF – Atrial Fibrillation
AS – Aortic Stenosis
MS – Mytral Stenosis
SC – Striato Capsular
DCSO – Discrete Centrum Semiovale
LCSO – Linear Centrum Semiolvae
Senso – Sensorium

LCR – Linear Corona Radiata
FSC – Frontal Subcortical
PSC – Parietal Subcortical
ABZ – Anterior Border Zone
PBZ – Posterior Border Zone
Patt – Pattern
L – Large
Lac – Lacunar

DCR – Discrete Corona Radiata	